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FILE 'HOME' ENTERED AT 06:46:00 ON 11 OCT 2008

=> fil .bio

FILE 'MEDLINE' ENTERED AT 06:46:10 ON 11 OCT 2008

FILE 'BIOSIS' ENTERED AT 06:46:10 ON 11 OCT 2008

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FILE 'EMBASE' ENTERED AT 06:46:10 ON 11 OCT 2008

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=> e sundrehagen e/au

E1	2	SUNDRE S M/AU
E2	1	SUNDRE STEVEN M/AU
E3	79 -->	SUNDREHAGEN E/AU
E4	79	SUNDREHAGEN ERLING/AU
E5	2	SUNDRESAN V/AU
E6	2	SUNDRESAN VASI/AU
E7	3	SUNDRI G/AU
E8	1	SUNDRI KRISHNA/AU
E9	1	SUNDRI R B T/AU
E10	1	SUNDRI SATTIRAJU KRISHNA/AU
E11	4	SUNDRIC Z/AU
E12	1	SUNDRIC Z S/AU

=> s e3-e4

L1 155 ("SUNDREHAGEN E"/AU OR "SUNDREHAGEN ERLING"/AU)

=> set linelength 250 perm

SET COMMAND COMPLETED

=> s calprotectin OR (l1(a)(antigen OR protein)) OR ((calcium OR calcium-binding)(2a)myeloid(2a)(8 OR 14 OR p8 OR p14)) OR mrp8 OR mrp-8 OR mrp14 OR mrp-14
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L1(A)(ANTIGEN'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L4(A)(ANTIGEN'
L2 2994 CALPROTECTIN OR (L1(A)(ANTIGEN OR PROTEIN)) OR ((CALCIUM OR CALCIUM-BINDING)(2A) MYELOID(2A)(8 OR 14 OR P8 OR P14)) OR MRP8 OR MRP-8 OR MRP14 OR MRP-14

=> s calprotectin OR ("l1"(a)(antigen OR protein)) OR ((calcium OR calcium-binding)(2a)myeloid(2a)(8 OR 14 OR p8 OR p14)) OR mrp8 OR mrp-8 OR mrp14 OR mrp-14
L3 7144 CALPROTECTIN OR ("L1"(A)(ANTIGEN OR PROTEIN)) OR ((CALCIUM OR CALCIUM-BINDING)(2A) MYELOID(2A)(8 OR 14 OR P8 OR P14)) OR MRP8 OR MRP-8 OR MRP14 OR MRP-14

=> s calgranulin OR (migratory(a)inhibitory(3a)protein) OR mif8 OR mif-8 OR mif-14 OR mif14 OR 27E10 OR L1H OR L1L OR S100A8 OR S100-A8 OR S100(a)A8 OR S100A9 OR S100-A9 OR S100(a)A9
L4 3693 CALGRANULIN OR (MIGRATORY(A) INHIBITORY(3A) PROTEIN) OR MIF8 OR MIF-

8 OR MIF-14 OR MIF14 OR 27E10 OR L1H OR L1L OR S100A8 OR S100-A8 OR S100(A) A8 OR
S100A9 OR S100-A9 OR S100(A) A9

=> s 13 OR 14
L5 9967 L3 OR L4

=> s L5(25a)(heart OR cvd OR acs OR coronary OR cardiac OR cardio? OR
cardiovascular OR atherosclero? OR arterioscler? OR myocard?)
L6 118 L5(25A)(HEART OR CVD OR ACS OR CORONARY OR CARDIAC OR CARDIO? OR
CARDIOVASCULAR OR ATHEROSCLERO? OR ARTERIOSCLER? OR MYOCARD?)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 68 DUP REM L6 (50 DUPLICATES REMOVED)

=> s l1 AND l5
L8 1 L1 AND L5

=> d ibib ed abs l8 1

L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:546624 CAPLUS Full-text
DOCUMENT NUMBER: 141:67871
TITLE: Cardiovascular disease assay
INVENTOR(S): Sundrehagen, Erling
PATENT ASSIGNEE(S): Axis-Shield Asa, Norway; Cockbain, Julian
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 2004057341	A2	20040708	WO 2003-GB5607	20031222
WO 2004057341	A3	20041021		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2504876	A1	20040708	CA 2003-2504876	20031222
AU 2003295142	A1	20040714	AU 2003-295142	20031222
EP 1573335	A2	20050914	EP 2003-786143	20031222
EP 1573335	B1	20071205		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1729398	A	20060201	CN 2003-80106912	20031222
JP 2006510895	T	20060330	JP 2004-561672	20031222
EP 1739430	A2	20070103	EP 2006-21890	20031222
EP 1739430	A3	20070117		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LI, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AT 380345	T	20071215	AT 2003-786143	20031222
ES 2297250	T3	20080501	ES 2003-786143	20031222

NO 2005002161	A	20050719	NO 2005-2161	20050503
US 20060134705	A1	20060622	US 2005-539797	20051219
PRIORITY APPLN. INFO.:			GB 2002-29747	A 20021220
			EP 2003-786143	A3 20031222
			WO 2003-GB5607	W 20031222

ED Entered STN: 08 Jul 2004

AB An assay method for the detection of potential for CVD or propensity to CVD in a human or non-human animal subject, said method comprising assessing the concentration of calprotectin in a calprotectin containing sample taken from said subject.

=> d ibib ed abs 17 1-68

L7 ANSWER 1 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:1114254 CAPLUS Full-text

DOCUMENT NUMBER: 149:328951

TITLE: Early release of S100A8/A9 Protein in Patients with Acute Coronary Syndromes

AUTHOR(S): Hammer, Florian

CORPORATE SOURCE: Germany

SOURCE: (2008) No pp. Avail.: Metadata on Internet Documents, Order No. 381240

From: Metadata Internet Doc. [Ger. Diss.] 2008, (D0916-1),

No pp. given

URL: <http://www.meind.de/search.py?recid=381240>

DOCUMENT TYPE: Dissertation

LANGUAGE: German

ED Entered STN: 17 Sep 2008

AB Unavailable

L7 ANSWER 2 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:555533 CAPLUS Full-text

DOCUMENT NUMBER: 148:515231

TITLE: A common gene expression signature in dilated cardiomyopathy and a microarray for diagnosis of the disease

INVENTOR(S): Kuner, Ruprecht; Sueltmann, Holger; Ruschhaupt, Markus; Bunes, Andreas; Barth, Andreas; Nabauer, Michael; Poustka, Annemarie

PATENT ASSIGNEE(S): Deutsches Krebsforschungszentrum, Germany

SOURCE: PCT Int. Appl., 53pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 2008053358	A2	20080508	WO 2007-IB4191	20070724
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA,				

GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ,
UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2006-832959P P 20060725

ED Entered STN: 09 May 2008

AB A small number of highly informative gene expression markers for dilated cardiomyopathy is identified. Expression of these genes can be detected and quantified using a microarray for rapid diagnosis of the disease. Gene expression profiling identified a panel of 76 genes showing changes in levels of expression in dilated cardiomyopathy, with 49 common to other forms of heart failure and 27 specific to dilated cardiomyopathy.

L7 ANSWER 3 OF 68 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2008125034 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 18252823

TITLE: Type 1 diabetes promotes disruption of advanced atherosclerotic lesions in LDL receptor-deficient mice.

AUTHOR: Johansson Fredrik; Kramer Farah; Barnhart Shelley; Kanter Jenny E; Vaisar Tomas; Merrill Rachel D; Geng Linda; Oka Kazuhiro; Chan Lawrence; Chait Alan; Heinecke Jay W; Bornfeldt Karin E

CORPORATE SOURCE: Department of Pathology, University of Washington, Seattle, WA 98195, USA.

CONTRACT NUMBER: DK002456 (United States NIDDK)
DK02456 (United States NIDDK)
HL076719 (United States NHLBI)
HL078527 (United States NHLBI)
HL088627 (United States NHLBI)
HL30086 (United States NHLBI)
HL51586 (United States NHLBI)
HL59314 (United States NHLBI)
HL62887 (United States NHLBI)
HL73144 (United States NHLBI)

SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (2008 Feb 12) Vol. 105, No. 6, pp. 2082-7. Electronic Publication: 2008-02-05.

Journal code: 7505876. E-ISSN: 1091-6490.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200803

ENTRY DATE: Entered STN: 21 Feb 2008

Last Updated on STN: 1 Apr 2008

Entered Medline: 31 Mar 2008

ED Entered STN: 21 Feb 2008

Last Updated on STN: 1 Apr 2008

Entered Medline: 31 Mar 2008

AB Cardiovascular disease, largely because of disruption of atherosclerotic lesions, accounts for the majority of deaths in people with type 1 diabetes. Recent mouse models have provided insights into the accelerated atherosclerotic lesion initiation in diabetes, but it is unknown whether diabetes directly worsens more clinically relevant advanced lesions. We therefore used an LDL receptor-deficient mouse model, in which type 1 diabetes can be induced at will, to investigate the effects of diabetes on preexisting lesions. Advanced lesions were induced by feeding mice a high-fat diet for 16 weeks before induction of diabetes. Diabetes, independently of lesion size, increased intraplaque hemorrhage and plaque disruption in the brachiocephalic artery of mice fed low-fat or high-fat diets for an additional 14 weeks.

Hyperglycemia was not sufficient to induce plaque disruption. Furthermore, diabetes resulted in increased accumulation of monocytic cells positive for S100A9, a proinflammatory biomarker for cardiovascular events, and for a macrophage marker protein, without increasing lesion macrophage content. S100A9 immunoreactivity correlated with intraplaque hemorrhage. Aggressive lowering primarily of triglyceride-rich lipoproteins prevented both plaque disruption and the increased S100A9 in diabetic atherosclerotic lesions. Conversely, oleate promoted macrophage differentiation into an S100A9-positive population in vitro, thereby mimicking the effects of diabetes. Thus, diabetes increases plaque disruption, independently of effects on plaque initiation, through a mechanism that requires triglyceride-rich lipoproteins and is associated with an increased accumulation of S100A9-positive monocytic cells. These findings indicate an important link between diabetes, plaque disruption, and the innate immune system.

L7 ANSWER 4 OF 68 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2008333422 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 18403730
TITLE: S100A8 and S100A9 mediate endotoxin-induced cardiomyocyte dysfunction via the receptor for advanced glycation end products.
AUTHOR: Boyd John H; Kan Bernard; Roberts Haley; Wang Yingjin; Walley Keith R
CORPORATE SOURCE: Critical Care Research Laboratories, St. Paul' Hospital, University of British Columbia, Vancouver, Canada.. jboyd@mrl.ubc.ca
SOURCE: Circulation research, (2008 May 23) Vol. 102, No. 10, pp. 1239-46. Electronic Publication: 2008-04-10.
Journal code: 0047103. E-ISSN: 1524-4571.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200806
ENTRY DATE: Entered STN: 24 May 2008
Last Updated on STN: 19 Jun 2008
Entered Medline: 17 Jun 2008
ED Entered STN: 24 May 2008
Last Updated on STN: 19 Jun 2008
Entered Medline: 17 Jun 2008
AB Cardiovascular dysfunction as a result of sepsis is the leading cause of death in the critically ill. Cardiomyocytes respond to infectious pathogens with a Toll-like receptor-initiated proinflammatory response in conjunction with a decrease in contractility, although the downstream events linking Toll-like receptor activation and reduced cardiac contractility remain to be elucidated. Using microarray analysis of cardiac tissue exposed to systemic lipopolysaccharide (LPS), we discovered that 2 small calcium-regulating proteins (S100A8 and S100A9) are highly upregulated. HL-1 cardiomyocytes, isolated primary cardiomyocytes, and live mice were exposed to LPS, whereas beating HL-1 cells had S100A8 and S100A9 overexpressed and their calcium flux quantified. Using in vivo microbubble technology, we delivered S100A8 and S100A9 to normal mouse hearts; using the same technology, we inhibited S100A9 production in mouse hearts and subsequently exposed them to LPS. Coimmunoprecipitation of S100A8 and S100A9 identified interaction with RAGE (the receptor for advanced glycation end products), the cardiac function and postreceptor signaling of which were investigated. HL-1 cardiomyocytes, isolated primary cardiomyocytes, and whole hearts exposed to LPS have large increases in S100A8 and S100A9. Cardiac overexpression of S100A8 and S100A9 led to a RAGE-dependent decrease in calcium flux and, in the intact mouse, to a decreased cardiac ejection fraction, whereas knockdown of S100A9 attenuated

LPS-induced cardiac dysfunction. Cardiomyocytes exposed to LPS express S100A8 and S100A9, leading to a RAGE-mediated decrease in cardiomyocyte contractility. This finding provides a novel mechanistic link between circulating pathogen-associated molecular products and subsequent cardiac dysfunction.

L7 ANSWER 5 OF 68 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2008445148 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 18308864
TITLE: Increased serum levels and expression of S100A8/A9 complex in infiltrated neutrophils in atherosclerotic plaque of unstable angina.
AUTHOR: Miyamoto S; Ueda M; Ikemoto M; Naruko T; Itoh A; Tamaki S; Nohara R; Terasaki F; Sasayama S; Fujita M
CORPORATE SOURCE: Division of Cardiology, Kitano Hospital, Tadukekofukai Medical Research Institute, Osaka, Japan.
SOURCE: Heart (British Cardiac Society), (2008 Aug) Vol. 94, No. 8, pp. 1002-7. Electronic Publication: 2008-02-28.
JOURNAL: Journal code: 9602087. E-ISSN: 1468-201X.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200808
ENTRY DATE: Entered STN: 16 Jul 2008
Last Updated on STN: 8 Aug 2008
Entered Medline: 7 Aug 2008
ED Entered STN: 16 Jul 2008
Last Updated on STN: 8 Aug 2008
Entered Medline: 7 Aug 2008
AB BACKGROUND: The S100A8/A9 complex is expressed in a subset of activated neutrophils and macrophages in acute inflammatory lesions associated with various diseases. OBJECTIVE: To investigate (a) whether serum S100A8/A9 levels are increased in patients with unstable angina (UA); and (b) whether S100A8/A9 expression is upregulated in coronary atherosclerotic plaques of patients with UA. DESIGN: Serum S100A8/A9 levels in 39 patients with stable angina (SA) and 53 patients with UA were measured. In addition, the presence of the S100A8/A9 complex in directional coronary atherectomy specimens was studied immunohistochemically. Cell types which stain positive for S100A8/A9 were identified by immunodouble staining with neutrophils and macrophages. RESULTS: Mean (SD) serum S100A8/A9 levels were significantly higher in patients with UA than in those with SA (3.25 (3.08) microg/ml vs 0.77 (0.31) microg/ml, p<0.05). In patients with UA, immunodouble staining clearly showed that the S100A8/A9 complex was expressed in infiltrated neutrophils and occasional macrophages. The S100A8/A9-positive area was significantly higher in UA than in SA (mean (SD) 18.3 (14.2)% vs 1.3 (2.4)%, respectively, p<0.001). CONCLUSIONS: The S100A8/A9 complex may be involved in the inflammatory process of coronary atherosclerotic plaques in patients with UA.

L7 ANSWER 6 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2008:680524 CAPLUS Full-text
DOCUMENT NUMBER: 149:220352
TITLE: Biomarkers and bioassays for cardiovascular diseases: present and future
AUTHOR(S): Sim, Derek S.; Lieu, Hsiao; Andre, Patrick
CORPORATE SOURCE: Department of Biology, Portola Pharmaceuticals Inc., South San Francisco, CA, USA
SOURCE: Biomarker Insights (2008) 293-302
CODEN: BIINHJ; ISSN: 1177-2719

URL: [http://la-](http://la-press.com/redirect_file.php?fileId=861&filename=BMI-3-Andre-et-al&fileType=pdf)

[press.com/redirect_file.php?fileId=861&filename=BMI-3-Andre-et-al&fileType=pdf](http://la-press.com/redirect_file.php?fileId=861&filename=BMI-3-Andre-et-al&fileType=pdf)

PUBLISHER: Libertas Academia Ltd.

DOCUMENT TYPE: Journal; General Review; (online computer file)

LANGUAGE: English

ED Entered STN: 06 Jun 2008

AB A review. Stratification of cardiac patients arriving at the emergency department is now being made according to the levels of acute cardiac biomarkers (i.e. cardiac troponin (cTn) or creatine kinase myocardial band (CK-MB)). Ongoing efforts are undertaken in an attempt to identify and validate additional cardiac biomarkers, for example, interleukin-6, soluble CD40L, and C-reactive protein, in order to further risk stratify patients with acute coronary syndrome. Several studies have also now shown an association of platelet transcriptome and genomic single nucleotide polymorphisms with myocardial infarction by using advanced genomic tools. A number of markers, such as myeloid-related protein 14 (MRP-14), cyclooxygenase-1 (COX-1), 5-lipoxygenase activating protein (FLAP), leukotriene A4 hydrolase (LTA4H) and myocyte enhancing factor 2A (MEF2A), have been linked to acute coronary syndromes, including myocardial infarction. In the future, these novel markers may pave the way toward personalized disease-prevention programs based on a person's genomic, thrombotic and cardiovascular profiles. Current and future biomarkers and bioassays for identifying at-risk patients will be discussed in this review.

REFERENCE COUNT: 92 THERE ARE 92 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 7 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:45765 CAPLUS Full-text

DOCUMENT NUMBER: 148:235931

TITLE: Vascular and inflammatory stresses mediate atherosclerosis via RAGE and its ligands in apoE-/- mice

AUTHOR(S): Harja, Evis; Bu, De-xiu; Hudson, Barry I.; Chang, Jong Sun; Shen, Xiaoping; Hallam, Kellie; Kalea, Anastasia Z.; Lu, Yan; Rosario, Rosa H.; Oruganti, Sai; Nikolla, Zana; Belov, Dmitri; Lalla, Evanthia; Ramasamy, Ravichandran; Yan, Shi Fang; Schmidt, Ann Marie

CORPORATE SOURCE: Division of Surgical Science, Department of Surgery, Columbia University Medical Center, New York, NY, USA

SOURCE: Journal of Clinical Investigation (2008), 118(1), 183-194
CODEN: JCINAO; ISSN: 0021-9738

PUBLISHER: American Society for Clinical Investigation

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 11 Jan 2008

AB Endothelial dysfunction is a key triggering event in atherosclerosis. Following the entry of lipoproteins into the vessel wall, their rapid modification results in the generation of advanced glycation endproduct epitopes and subsequent infiltration of inflammatory cells. These inflammatory cells release receptor for advanced glycation endproduct (RAGE) ligands, specifically S100/calgranulins and high-mobility group box 1, which sustain vascular injury. Here, we demonstrate critical roles for RAGE and its ligands in vascular inflammation, endothelial dysfunction, and atherosclerotic plaque development in a mouse model of atherosclerosis, apoE-/- mice. Expts. in primary aortic endothelial cells isolated from mice and in cultured human aortic endothelial cells revealed the central role of JNK signaling in transducing the impact of RAGE ligands on inflammation. These data highlight unifying mechanisms whereby endothelial RAGE and its ligands mediate vascular and inflammatory stresses that culminate in atherosclerosis in the vulnerable vessel wall.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 8 OF 68 MEDLINE on STN DUPLICATE 5

ACCESSION NUMBER: 2007746037 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 18082488

TITLE: Myeloid-related protein 8/14 and the risk of cardiovascular death or myocardial infarction after an acute coronary syndrome in the Pravastatin or Atorvastatin Evaluation and Infection Therapy: Thrombolysis in Myocardial Infarction (PROVE IT-TIMI 22) trial.

AUTHOR: Morrow David A; Wang Yunmei; Croce Kevin; Sakuma Masashi; Sabatine Marc S; Gao Huiyun; Pradhan Aruna D; Healy Aileen M; Burows Jacki; McCabe Carolyn H; Libby Peter; Cannon Christopher P; Braunwald Eugene; Simon Daniel I

CORPORATE SOURCE: Thrombolysis in Myocardial Infarction Study Group, Brigham and Women's Hospital, Boston, MA 02115, USA.. dmorrow@rics.bwh.harvard.edu

CONTRACT NUMBER: HL082740 (United States NHLBI)
HL57506 (United States NHLBI)
HL60942 (United States NHLBI)
U01 HL083-1341 (United States NHLBI)

SOURCE: American heart journal, (2008 Jan) Vol. 155, No. 1, pp. 49-55.

Electronic Publication: 2007-11-01.

Journal code: 0370465. E-ISSN: 1097-6744.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(CLINICAL TRIAL)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200801

ENTRY DATE: Entered STN: 18 Dec 2007

Last Updated on STN: 12 Jan 2008

Entered Medline: 10 Jan 2008

ED Entered STN: 18 Dec 2007

Last Updated on STN: 12 Jan 2008

Entered Medline: 10 Jan 2008

AB BACKGROUND: Using a transcriptional profiling approach, we recently identified myeloid-related protein 8/14 (MRP-8/14) to be expressed by platelets during acute myocardial infarction (MI). Elevated concentrations of MRP-8/14 are associated with a higher risk for future cardiovascular events in apparently healthy individuals but have not been assessed with respect to prognosis in patients with acute coronary syndrome. METHODS: We performed a nested case-control study (n = 237 case-control pairs) among patients enrolled in the Pravastatin or Atorvastatin Evaluation and Infection Therapy: Thrombolysis in Myocardial Infarction 22 (PROVE IT-TIMI 22) trial (mean follow-up 24 months) to investigate the risk of cardiovascular death or MI associated with MRP-8/14 measured at 30 days after an acute coronary syndrome. RESULTS: Patients with cardiovascular death or MI after 30 days (cases) had higher median [25th, 75th percentile] MRP-8/14 levels than patients who remained free of recurrent events (5.6 [2.8, 13.5] mg/L vs 4.0 [1.9, 10.1] mg/L, P = .020). The risk of a recurrent cardiovascular event increased with each increasing quartile of MRP-8/14 (P-trend = 0.007) such that patients with the highest levels had a 2.0-fold increased odds (95% CI 1.1-3.6, P = .029) of a recurrent event after adjusting for standard risk indicators, randomized treatment, and C-reactive protein. Patients with elevated levels of MRP-8/14 and high-sensitivity C-reactive protein showed significantly increased risk of cardiovascular death or MI compared with patients with the lowest levels of both markers (adjusted odds ratio 2.1, 95% CI 1.2-3.8). CONCLUSIONS: Myeloid-related protein 8/14 may be a useful biomarker of platelet and inflammatory disease activity in atherothrombosis and may serve as a novel target for therapeutic intervention.

L7 ANSWER 9 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:147132 CAPLUS Full-text

TITLE: Type 1 diabetes promotes disruption of advanced atherosclerotic lesions in LDL receptor-deficient mice

AUTHOR(S): Johansson, Fredrik; Kramer, Farah; Barnhart, Shelley; Kanter, Jenny E.; Vaisar, Tomas; Merrill, Rachel D.; Geng, Linda; Oka, Kazuhiro; Chan, Lawrence; Chait, Alan; Heinecke, Jay W.; Bornfeldt, Karin E.

CORPORATE SOURCE: Department of Pathology, University of Washington, Seattle, WA, 98195, USA

SOURCE: Proceedings of the National Academy of Sciences of the United States of America, Early Edition (2008), (Feb 5 2008), 1-6, 6 pp.

CODEN: PNASC8

URL: <http://www.pnas.org/cgi/reprint/0709958105v1>

PUBLISHER: National Academy of Sciences

DOCUMENT TYPE: Journal; (online computer file)

LANGUAGE: English

ED Entered STN: 06 Feb 2008

AB Cardiovascular disease, largely because of disruption of atherosclerotic lesions, accounts for the majority of deaths in people with type 1 diabetes. Recent mouse models have provided insights into the accelerated atherosclerotic lesion initiation in diabetes, but it is unknown whether diabetes directly worsens more clin. relevant advanced lesions. We therefore used an LDL receptor-deficient mouse model, in which type 1 diabetes can be induced at will, to investigate the effects of diabetes on preexisting lesions. Advanced lesions were induced by feeding mice a high-fat diet for 16 wk before induction of diabetes. Diabetes, independently of lesion size, increased intraplaque hemorrhage and plaque disruption in the brachiocephalic artery of mice fed low-fat or high-fat diets for an addnl. 14 wk. Hyperglycemia was not sufficient to induce plaque disruption. Furthermore, diabetes resulted in increased accumulation of monocytic cells pos. for S100A9, a proinflammatory biomarker for cardiovascular events, and for a macrophage marker protein, without increasing lesion macrophage content. S100A9 immunoreactivity correlated with intraplaque hemorrhage. Aggressive lowering primarily of triglyceride-rich lipoproteins prevented both plaque disruption and the increased S100A9 in diabetic atherosclerotic lesions. Conversely, oleate promoted macrophage differentiation into an S100A9-pos. population in vitro, thereby mimicking the effects of diabetes. Thus, diabetes increases plaque disruption, independently of effects on plaque initiation, through a mechanism that requires triglyceride-rich lipoproteins and is associated with an increased accumulation of S100A9-pos. monocytic cells. These findings indicate an important link between diabetes, plaque disruption, and the innate immune system.

L7 ANSWER 10 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:732306 CAPLUS Full-text

DOCUMENT NUMBER: 149:29490

TITLE: Early release of S100A8/A9 Protein in Patients with Acute Coronary Syndromes

AUTHOR(S): Hammer, Florian

CORPORATE SOURCE: Germany

SOURCE: (2007) No pp. Avail.: Metadata on Internet Documents, Order No. 381240

From: Metadata Internet Doc. [Ger. Diss.] 2007, (D0617-1),

No pp. given

URL: <http://www.meind.de/search.py?recid=381240>

DOCUMENT TYPE: Dissertation

LANGUAGE: German

ED Entered STN: 19 Jun 2008

AB Unavailable

L7 ANSWER 11 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:1391017 CAPLUS Full-text
DOCUMENT NUMBER: 147:539665
TITLE: The use of MRP 8/14 levels for discrimination of
individuals at risk of acute coronary syndromes
INVENTOR(S): Maier, Willibald; Altwegg, Lukas; Hersberger, Martin;
Neidhart, Michel
PATENT ASSIGNEE(S): Roche Diagnostics G.m.b.H., Germany; F.Hoffmann-La Roche
A.-G.
SOURCE: PCT Int. Appl., 55pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2007137865	A1	20071206	WO 2007-EP4872	20070601
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM EP 1862805 A1 20071205 EP 2006-11416 20060601 R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU PRIORITY APPLN. INFO.: EP 2006-11416 A 20060601 US 2006-803642P P 20060601				

ED Entered STN: 06 Dec 2007

AB The present invention relates to a method for determining the risk whether an individual showing at least one symptom of an evolving acute coronary syndrome (ACS) is suffering from an acute coronary syndrome comprising the steps of (a) measuring, preferably in vitro, the level of MRP 8/14, wherein (b) if the level of MRP 8/14 is at least increased, then the individual is at risk of suffering from an acute coronary syndrome. The invention further relates to methods for ruling out whether an individual showing at least one symptom of an evolving ACS is suffering from an ACS, for assessing whether an individual showing at least one symptom of an evolving ACS is not at risk of suffering from an ACS and to discriminate if an individual showing at least one symptom of an evolving ACS is at risk to suffer from an ACS and or has no ACS.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 12 OF 68 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2007573802 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 17895549
TITLE: Enhanced expression of myeloid-related protein complex
(MRP8/14) in macrophages and multinucleated giant cells in granulomas of patients with active cardiac sarcoidosis.
AUTHOR: Terasaki Fumio; Fujita Masatoshi; Shimomura Hiroaki; Tsukada Bin; Otsuka Koji; Otsuka Kaoru; Katashima Takashi; Ikemoto Masaki; Kitaura Yasushi

CORPORATE SOURCE: Department of Internal Medicine III, Osaka Medical College, 2-7 Daigaku-machi, Takatsuki 569-8686, Japan.. in3012@poh.osaka-med.ac.jp
SOURCE: Circulation journal : official journal of the Japanese Circulation Society, (2007 Oct) Vol. 71, No. 10, pp. 1545-50.
Journal code: 101137683. ISSN: 1346-9843.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200712
ENTRY DATE: Entered STN: 27 Sep 2007
Last Updated on STN: 11 Dec 2007
Entered Medline: 7 Dec 2007

ED Entered STN: 27 Sep 2007
Last Updated on STN: 11 Dec 2007
Entered Medline: 7 Dec 2007

AB BACKGROUND: The myeloid-related protein complex (MRP8/14) is expressed in activated human macrophages and reported to be involved in the inflammatory process. The expression of MRP8/14 in patients with cardiac sarcoidosis and idiopathic dilated cardiomyopathy (DCM) was investigated. METHODS AND RESULTS: Serum MRP8/14 levels were measured in 35 patients with sarcoidosis and 23 patients with DCM. Sera from 30 normal volunteers served as controls. Additionally, the expression profiles of MRP8/14 in the myocardium from 12 patients with active cardiac sarcoidosis and 10 DCM patients were examined immunohistochemically. Serum MRP8/14 levels were significantly higher in patients with sarcoidosis than in normal controls [515+/-549 (SD) ng/ml vs 230+/-115 ng/ml, p=0.0019]. In the sarcoidosis group, serum MRP8/14 levels in patients with definite cardiac involvement (n=10) were significantly higher than in those without (n=25) (974+/-878 ng/ml vs 332+/-204 ng/ml, p=0.0227) and they were also higher than in DCM patients (vs 252+/-108 ng/ml, p=0.0026). Immunohistochemically, MRP8/14 was specifically positive in the cytoplasm of macrophages and multinucleated giant cells in the myocardial granulomas. CONCLUSIONS: MRP8/14 may be involved in the pathogenesis of sarcoid granulomas. The measurement of serum MRP8/14 levels is useful for the diagnosis of sarcoidosis, and their higher levels suggest the cardiac involvement.

L7 ANSWER 13 OF 68 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2007615464 EMBASE Full-text
TITLE: Application and establishment of two-dimensional gel electrophoresis from proteome analysis of human osteosarcoma.
AUTHOR: Yang, Xiao-Yu (correspondence); Gu, Rui; Gao, Zhong-Li; Yin, Zhao-Yang; Zhang, Shan-Yong
CORPORATE SOURCE: Department of Orthopaedics, China-Japan Union Hospital, Jilin University, Changchun 130033, China. yxy5649255@sina.com
AUTHOR: Sun, Wei
CORPORATE SOURCE: Proteomics Research Center, Institute of Basic Medical Sciences, Peking Union Medical College, Beijing 100005, China.
AUTHOR: Sui, Fu-Ge
SOURCE: Journal of Jilin University Medicine Edition, (28 Nov 2007)
Vol. 33, No. 6, pp. 1038-1042.
Refs: 12
ISSN: 1671-587X CODEN: JDXYA3
COUNTRY: China
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
027 Biophysics, Bioengineering and Medical Instrumentation

029 Clinical and Experimental Biochemistry
033 Orthopedic Surgery

LANGUAGE: Chinese
SUMMARY LANGUAGE: Chinese; English
ENTRY DATE: Entered STN: 9 Jan 2008
Last Updated on STN: 9 Jan 2008

ED Entered STN: 9 Jan 2008

Last Updated on STN: 9 Jan 2008

AB Objective: To establish analysis methods of two-dimensional (2-DE) gel electrophoresis for human osteosarcoma. Methods: A series of methods, including Immobilized pH gradient were used as ID. Some applications, such as sample preparation used as choice of IPG gel, were improved. Coomassie brilliant blue staining, ImageMaster 2D Elite 3.01 analysis software, MALDI-TOF/TOF MS and SWISS-PROT database searching were used to separate and indentify the proteins of human osteosarcoma. Results: The good use pattern including repetitive experiments showed that in three experiments, the amount of protein spots of the same team sample deviates from the relative standard as following, the average of variation coefficient (%): 23.00 ± 10.11 and 20.33 ± 9.90 ; and the range of variation coefficient (%) were: 3.80 - 6.89 and 2.70 - 6.89 from osteosarcoma and normal group respectively. The isoelectric point and molecular weigh of the same protein spots in three experiments deviated from relative standard as following: $(8.93 \pm 1.17)\%$, $(10.16 \pm 2.02)\%$, $(10.87 \pm 3.86)\%$, respectively. Therefore, better resolution and repetitive 2-DE atlas were obtained. The proteins from 11 pairs of sample were analysed by mass spectrometry, 9 identified proteins (transthyretin precursor, Triosephosphate isomerase, slow skeletal muscle, cardiac muscle Troponin T, Cofilin-1, Myosin light chain 1, Calgranulin B, Heat-shock protein, Annexin A5, Fanconi anemia group D2 proteins) were more abundant in osteosarcoma tisstues and 2 proteins manganese SOD and carbonic dehydratase appeared down-regulation in osteosarcoma tisstues. Conclusion: This optimized 2-DE map is an important tool for further study on osteosarcoma, and these identified proteins were related -proteins with osteosarcoma. It is suggested that the changes of the proteome are involved in the pathology processe of osteosarcoma.

L7 ANSWER 14 OF 68 MEDLINE on STN DUPLICATE 7

ACCESSION NUMBER: 2007235402 MEDLINE [Full-text](#)

DOCUMENT NUMBER: PubMed ID: 17387139

TITLE: Myeloid-related protein 8/14 complex is released by monocytes and granulocytes at the site of coronary occlusion: a novel, early, and sensitive marker of acute coronary syndromes.

AUTHOR: Altwegg Lukas A; Neidhart Michel; Hersberger Martin; Muller Simone; Eberli Franz R; Corti Roberto; Roffi Marco; Sutsch Gabor; Gay Steffen; von Eckardstein Arnold; Wischnowsky Manfred B; Luscher Thomas F; Maier Willibald
CORPORATE SOURCE: Cardiovascular Center, Cardiology, University Hospital Zurich, Ramistrasse 100, CH-8091 Zurich, Switzerland.

SOURCE: European heart journal, (2007 Apr) Vol. 28, No. 8, pp. 941-8.
Electronic Publication: 2007-03-26.

Journal code: 8006263. ISSN: 0195-668X.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200711

ENTRY DATE: Entered STN: 21 Apr 2007

Last Updated on STN: 8 Dec 2007

Entered Medline: 28 Nov 2007

ED Entered STN: 21 Apr 2007

Last Updated on STN: 8 Dec 2007

Entered Medline: 28 Nov 2007

AB AIMS: We investigated whether myeloid-related protein 8/14 complex (MRP8/14) expressed by infiltrating monocytes and granulocytes may represent a mediator and early biomarker of acute coronary syndromes (ACS). METHODS AND RESULTS: Immunohistochemistry of coronary thrombi was done in 41 ACS patients. Subsequently, levels of MRP8/14 were assessed systemically in 75 patients with ACS and culprit lesions, with stable coronary artery disease (CAD), or with normal coronary arteries. In a subset of patients, MRP8/14 was measured systemically and at the site of coronary occlusion. Macrophages and granulocytes, but not platelets stained positive for MRP8/14 in 76% of 41 thrombi patients. In ACS, local MRP8/14 levels [22.0 (16.2-41.5) mg/L] were increased when compared with systemic levels [13.4 (8.1-14.7) mg/L, P = 0.03]. Systemic levels of MRP8 /14 were markedly elevated [15.1 (12.1-21.8) mg/L, P = 0.001] in ACS when compared with stable CAD [4.6 (3.5-7.1) mg/L] or normals [4.8 (4.0-6.3) mg/L]. Using a cut-off level of 8 mg/L, MRP8/14 but not myoglobin or troponin, identified ACS presenting within 3 h from symptom onset. CONCLUSION: In ACS, MRP8/14 is markedly expressed at the site of coronary occlusion by invading phagocytes. The occurrence of elevated MRP8/14 in the systemic circulation prior to markers of myocardial necrosis makes it a prime candidate for the detection of unstable plaques and management of ACS.

L7 ANSWER 15 OF 68 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2007730959 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 18066133
TITLE: Clinical safety and efficacy of NG440: a novel combination of rho iso-alpha acids from hops, rosemary, and oleanolic acid for inflammatory conditions.
AUTHOR: Minich Deanna M; Bland Jeffrey S; Katke Jeffrey; Darland Gary; Hall Amy; Lerman Robert H; Lamb Joseph; Carroll Brian; Tripp Matthew
CORPORATE SOURCE: Functional Medicine Research Center, division of MetaProteomics, LLC., 9770 44th Avenue NW, Suite 100, Gig Harbor, WA 98332, USA.. deannaminich@metagenics.com
SOURCE: Canadian journal of physiology and pharmacology, (2007 Sep) Vol. 85, No. 9, pp. 872-83.
Journal code: 0372712. ISSN: 0008-4212.
PUB. COUNTRY: Canada
DOCUMENT TYPE: (CLINICAL TRIAL)
(Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200802
ENTRY DATE: Entered STN: 11 Dec 2007
Last Updated on STN: 16 Feb 2008
Entered Medline: 15 Feb 2008

ED Entered STN: 11 Dec 2007
Last Updated on STN: 16 Feb 2008
Entered Medline: 15 Feb 2008

AB In this report, we examine the clinical safety and efficacy of NG440, a phytochemical-based antiinflammatory formula consisting of a combination of rho iso-alpha acids from hops, rosemary, and oleanolic acid. In a previous study, we demonstrated that NG440 significantly decreased pain by 50% in patients with osteoarthritis. Consistent with these data, results from a multicentre trial indicate that NG440 reduced pain scores in patients with joint discomfort, as measured by VAS (visual analog scale) methodology. As demonstrated in an ex vivo clinical study, these effects on pain relief may be due to reduced inflammatory cytokine production including lower prostaglandin

E2 formation. Finally, strong data exist to suggest that NG440 is a safe formula for human consumption. Animal toxicity data revealed no adverse effects of NG440 at dosages ≤ 250 mg.kg⁻¹.day⁻¹ for 21 days. Furthermore, human trial data suggest that NG440 does not negatively impact cardiovascular and gastrointestinal markers normally affected by selective COX-2 enzyme inhibitors, including platelet function, blood pressure, blood cell count, or fecal calprotectin, a measure of gastrointestinal injury. In conclusion, NG440 may serve as a safe and efficacious alternative in some areas where specific COX-2 inhibitors have been traditionally used.

L7 ANSWER 16 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:199284 CAPLUS Full-text

DOCUMENT NUMBER: 148:259198

TITLE: Receptor for advanced glycation endproducts (RAGE): a formidable force in the pathogenesis of the cardiovascular complications of diabetes & aging

AUTHOR(S): Yan, Shi Fang; D'Agati, Vivette; Schmidt, Ann Marie; Ramasamy, Ravichandran

CORPORATE SOURCE: Department of Surgery, Columbia University Medical Center, New York, NY, 10032, USA

SOURCE: Current Molecular Medicine (2007), 7(8), 699-710
CODEN: CMMUBP; ISSN: 1566-5240

PUBLISHER: Bentham Science Publishers Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

ED Entered STN: 18 Feb 2008

AB A review. Unifying mechanisms for the consequences of aging and chronic diabetes are coming to light with the identification that common to both settings is the production and accumulation of the largely irreversible Advanced Glycation Endproducts (AGEs). AGEs impart multiple consequences in the tissues; a key means by which they exert maladaptive effects is via their interaction with and activation of their chief cell surface receptor, Receptor for AGE or RAGE. Although the time course, rate and extent of AGE generation and accumulation in diabetes and aging may be distinct, unifying outcomes of the ligand-RAGE interaction in the vasculature and heart are linked to upregulation of inflammatory and tissue-destructive mechanisms. Consistent with these concepts, administration of the ligand-binding decoy of RAGE, soluble or sRAGE, suppresses early initiation and progression of atherosclerosis in diabetic mice; suppresses exaggerated neointimal expansion consequent to arterial injury; and mitigates the adverse impact of ischemia/reperfusion injury in the heart. Importantly, the RAGE ligand repertoire upregulated in these settings is not limited to AGEs. The key finding that RAGE was a multi-ligand receptor unified the concept that in diabetes and aging, innate and adaptive inflammatory mechanisms contribute to the pathogenesis of tissue injury. We conclude that antagonism of RAGE may reflect a novel and therapeutically logical and safe target in cardiovascular stress induced by aging and chronic diabetes.

REFERENCE COUNT: 101 THERE ARE 101 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 17 OF 68 MEDLINE on STN

DUPLICATE 9

ACCESSION NUMBER: 2007652361 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 17636430

TITLE: S100A8/S100A9 and their association with cartilage and bone.

AUTHOR: Zreiqat H; Howlett C R; Gronthos S; Hume D; Geczy C L

CORPORATE SOURCE: Biomaterials and Tissue Engineering Research Unit, Biomedical Engineering, School of AMME, The University of Sydney, Sydney, NSW, Australia..
hzreiqat@usyd.edu.au

SOURCE: Journal of molecular histology, (2007 Oct) Vol. 38, No. 5, pp.

381-91. Electronic Publication: 2007-07-17.
Journal code: 101193653. ISSN: 1567-2379.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200803
ENTRY DATE: Entered STN: 6 Nov 2007
Last Updated on STN: 18 Mar 2008
Entered Medline: 17 Mar 2008

ED Entered STN: 6 Nov 2007
Last Updated on STN: 18 Mar 2008
Entered Medline: 17 Mar 2008

AB S100A8 and S100A9 are calcium-binding proteins expressed in myeloid cells and are markers of numerous inflammatory diseases in humans. S100A9 has been associated with dystrophic calcification in human atherosclerosis. Here we demonstrate S100A8 and S100A9 expression in murine and human bone and cartilage cells. Only S100A8 was seen in preosteogenic cells whereas osteoblasts had variable, but generally weak expression of both proteins. In keeping with their reported high-mRNA expression, S100A8 and S100A9 were prominent in osteoclasts. S100A8 was expressed in alkaline phosphatase-positive hypertrophic chondrocytes, but not in proliferating chondrocytes within the growth plate where the cartilaginous matrix was calcifying. S100A9 was only evident in the invading vascular osteogenic tissue penetrating the degenerating chondrocytic zone adjacent to the primary spongiosa, where S100A8 was also expressed. Whilst, S100A8 has been shown to be associated with osteoblast differentiation, both S100A8 and S100A9 may contribute to calcification of the cartilage matrix and its replacement with trabecular bone, and to regulation of redox in bone resorption.

L7 ANSWER 18 OF 68 MEDLINE on STN
ACCESSION NUMBER: 2007760952 IN-PROCESS Full-text
DOCUMENT NUMBER: PubMed ID: 17949489
TITLE: Effects of iron loading on muscle: genome-wide mRNA expression profiling in the mouse.
AUTHOR: Rodriguez Alejandra; Hilvo Mika; Kytomaki Leena; Fleming Robert E; Britton Robert S; Bacon Bruce R; Parkkila Seppo
CORPORATE SOURCE: Institute of Medical Technology, University of Tampere and Tampere University Hospital, Tampere, Finland.. alejandra.rodriguez.martinez@uta.fi
SOURCE: BMC genomics, (2007) Vol. 8, pp. 379. Electronic Publication: 2007-10-19.

Journal code: 100965258. E-ISSN: 1471-2164.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-DATA-REVIEW; IN-PROCESS; NONINDEXED
ENTRY DATE: Entered STN: 27 Dec 2007
Last Updated on STN: 27 Dec 2007

ED Entered STN: 27 Dec 2007
Last Updated on STN: 27 Dec 2007

AB ABSTRACT: BACKGROUND: Hereditary hemochromatosis (HH) encompasses genetic disorders of iron overload characterized by deficient expression or function of the iron-regulatory hormone hepcidin. Mutations in 5 genes have been linked to this disease: HFE, TFR2 (encoding transferrin receptor 2), HAMP (encoding hepcidin), SLC40A1 (encoding ferroportin) and HJV (encoding hemojuvelin). Hepcidin inhibits iron export from cells into plasma. Hemojuvelin, an upstream regulator of hepcidin expression, is expressed in mice mainly in the heart and skeletal muscle. It has been suggested that soluble hemojuvelin shed by the muscle might reach the liver to influence

hepcidin expression. Heart muscle is one of the target tissues affected by iron overload, with resultant cardiomyopathy in some HH patients. Therefore, we investigated the effect of iron overload on gene expression in skeletal muscle and heart using Illumina microarray arrays containing over 47,000 probes. The most apparent changes in gene expression were confirmed using real-time RT-PCR. RESULTS: Genes with up-regulated expression after iron overload in both skeletal and heart muscle included angiopoietin-like 4, pyruvate dehydrogenase kinase 4 and calgranulin A and B. The expression of transferrin receptor, heat shock protein 1B and DnaJ homolog B1 were down-regulated by iron in both muscle types. Two potential hepcidin regulatory genes, hemojuvelin and neogenin, showed no clear change in expression after iron overload. CONCLUSION: Microarray analysis revealed iron-induced changes in the expression of several genes involved in the regulation of glucose and lipid metabolism, transcription and cellular stress responses. These may represent novel connections between iron overload and pathological manifestations of HH such as cardiomyopathy and diabetes.

L7 ANSWER 19 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:238416 BIOSIS Full-text
DOCUMENT NUMBER: PREV200700240919
TITLE: Balance between serum S100A12 and S100A8 predicts presence and complications of coronary artery disease.
AUTHOR(S): Yan, Wei Xing [Reprint Author]; Song, Changjie; Yamen, Eric; Freedman, Saul B.; Geczy, Carolyn L.
CORPORATE SOURCE: Univ New S Wales, Sydney, NSW, Australia
SOURCE: Journal of the American College of Cardiology, (MAR 6 2007) Vol. 49, No. 9, Suppl. A, pp. 359A.
Meeting Info.: 56th Annual Scientific Session of the American College-of-Cardiology. New Orleans, LA, USA. March 24 -27, 2007. Amer Coll Cardiol. CODEN: JACCDI. ISSN: 0735-1097.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Apr 2007
Last Updated on STN: 11 Apr 2007
ED Entered STN: 11 Apr 2007
Last Updated on STN: 11 Apr 2007

L7 ANSWER 20 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:350178 CAPLUS Full-text
DOCUMENT NUMBER: 147:499819
TITLE: The RAGE connection to diabetes and atherosclerosis: an intertwined web of advanced glycation and inflammation
AUTHOR(S): Ramasamy, Ravichandran; Yan, Shi Fang; Schmidt, Ann Marie
CORPORATE SOURCE: Department of Surgery, Columbia University Medical Center, New York, NY, 10032, USA
SOURCE: Future Lipidology (2007), 2(2), 239-250
CODEN: FLUIBL; ISSN: 1746-0875
PUBLISHER: Future Medicine Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
ED Entered STN: 29 Mar 2007
AB A review. The receptor for advanced glycation end products (RAGE) transduces the impact of the products of nonenzymic glycation and oxidation of proteins, the advanced glycation end products, proinflammatory ligands S100/calgranulins and high mobility group 1. The ligand families of RAGE accumulate in the vasculature in diabetes and are enriched in atherosclerotic lesions, both in human and animal models. Experimentation in animal models of both Type 1 and 2 diabetes reveals that antagonism of the ligand-RAGE axis suppresses the

development and progression of vascular and inflammatory cell perturbation in the diabetic milieu; key processes linked to acceleration of atherosclerosis and exaggerated neointimal expansion consequent to arterial injury. We propose that blockade of RAGE may represent an effective target for therapeutic intervention in diabetes and its cardiovascular complications.

REFERENCE COUNT: 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 21 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 10

ACCESSION NUMBER: 2008:133147 BIOSIS Full-text

DOCUMENT NUMBER: PREV200800124739

TITLE: Effects of iron loading on muscle: genome-wide mRNA expression profiling in the mouse.

AUTHOR(S): Rodriguez, Alejandra [Reprint Author]; Hilvo, Mika; Kytomaki, Leena; Fleming, Robert E.; Britton, Robert S.; Bacon, Bruce R.; Parkkila, Seppo
CORPORATE SOURCE: Tampere Univ, Tampere Univ Hosp, Inst Med Technol, Tampere, Finland

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leena.kytomaki@btk.fi; flemingr@slu.edu; brittonr@slu.edu; baconbr@slu.edu;
seppo.parkkila@uta.fi

SOURCE: BMC Genomics, (OCT 19 2007) Vol. 8, pp. Article No.: 379.
ISSN: 1471-2164.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 20 Feb 2008

Last Updated on STN: 20 Feb 2008

ED Entered STN: 20 Feb 2008

Last Updated on STN: 20 Feb 2008

AB Background: Hereditary hemochromatosis (HH) encompasses genetic disorders of iron overload characterized by deficient expression or function of the iron-regulatory hormone hepcidin. Mutations in 5 genes have been linked to this disease: HFE, TFR2 (encoding transferrin receptor 2), HAMP (encoding hepcidin), SLC40AI (encoding ferroportin) and HJV (encoding hemojuvelin). Hepcidin inhibits iron export from cells into plasma. Hemojuvelin, an upstream regulator of hepcidin expression, is expressed in mice mainly in the heart and skeletal muscle. It has been suggested that soluble hemojuvelin shed by the muscle might reach the liver to influence hepcidin expression. Heart muscle is one of the target tissues affected by iron overload, with resultant cardiomyopathy in some HH patients. Therefore, we investigated the effect of iron overload on gene expression in skeletal muscle and heart using Illumina(TM) arrays containing over 47,000 probes. The most apparent changes in gene expression were confirmed using real-time RT-PCR. Results: Genes with up-regulated expression after iron overload in both skeletal and heart muscle included angiopoietin-like 4, pyruvate dehydrogenase kinase 4 and calgranulin A and B. The expression of transferrin receptor, heat shock protein 1B and DnaJ homolog B1 were down-regulated by iron in both muscle types. Two potential hepcidin regulatory genes, hemojuvelin and neogenin, showed no clear change in expression after iron overload. Conclusion: Microarray analysis revealed iron-induced changes in the expression of several genes involved in the regulation of glucose and lipid metabolism, transcription and cellular stress responses. These may represent novel connections between iron overload and pathological manifestations of HH such as cardiomyopathy and diabetes.

L7 ANSWER 22 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:978103 CAPLUS Full-text

DOCUMENT NUMBER: 145:333310

TITLE: Gene expression profiling in the identification of the etiology of heart failure

INVENTOR(S): Kittleson, Michelle; Hare, Joshua
 PATENT ASSIGNEE(S): USA
 SOURCE: PCT Int. Appl., 74pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006099336	A2	20060921	WO 2006-US8966	20060310
WO 2006099336	A3	20070510		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

US 20060246484 A1 20061102 US 2006-373812 20060310

PRIORITY APPLN. INFO.: US 2005-660370P P 20050310

ED Entered STN: 21 Sep 2006

AB Genes showing changes in levels of expression in the heart in ischemic and non-ischemic heart disease in comparison to the healthy heart are identified by DNA microarray technol. The data show different gene expression profiles in the two diseases, indicating different etiologies for heart failure arising from them.

L7 ANSWER 23 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:439979 CAPLUS Full-text

DOCUMENT NUMBER: 144:460796

TITLE: Use of calgranulin A and B in the promotion and inhibition of mineralized tissue formation

INVENTOR(S): Geczy, Carolyn L.; Zreiqat, Hala

PATENT ASSIGNEE(S): Newsouth Innovations Pty Limited, Australia

SOURCE: PCT Int. Appl., 59 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006047820	A1	20060511	WO 2005-AU1681	20051101
WO 2006047820	A9	20060713		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG,

ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
PRIORITY APPLN. INFO.: AU 2004-906284 A 20041101
ED Entered STN: 11 May 2006

AB The present invention provides a method for promoting the formation of mineralized tissue in a vertebrate, the method comprising administering to the vertebrate an effective amount of one or more of the following: a calgranulin A (S100A8) polypeptide; a calgranulin B (S100A9) polypeptide; a polynucleotide encoding S100A8 operably linked to a promoter; or a polynucleotide encoding S100A9 operably linked to a promoter. The present invention also provides a method for the treatment or prevention of a bone disorder in a vertebrate, the method comprising administering to the vertebrate an effective amount of one or more of the following: an S100A8 polypeptide; an S100A9 polypeptide; a polynucleotide encoding S100A8 operably linked to a promoter; or a polynucleotide encoding S100A9 operably linked to a promoter.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 24 OF 68 MEDLINE on STN DUPLICATE 11

ACCESSION NUMBER: 2006272028 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 16682612

TITLE: Platelet expression profiling and clinical validation of myeloid-related protein-14 as a novel determinant of cardiovascular events.

AUTHOR: Healy Aileen M; Pickard Michael D; Pradhan Aruna D; Wang Yunmei; Chen Zhiping; Croce Kevin; Sakuma Masashi; Shi Can; Zago Alexandre C; Garasic Joseph; Damokosh Andrew I; Dowie Tracy L; Poisson Louis; Lillie James; Libby Peter;

Ridker Paul M; Simon Daniel I

CORPORATE SOURCE: Millennium Pharmaceuticals, Inc, Cambridge, MA, USA.

CONTRACT NUMBER: HL57506 (United States NHLBI)

HL60942 (United States NHLBI)

SOURCE: Circulation, (2006 May 16) Vol. 113, No. 19, pp. 2278-84.

Electronic Publication: 2006-05-08.

Journal code: 0147763. E-ISSN: 1524-4539.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(VALIDATION STUDIES)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200606

ENTRY DATE: Entered STN: 17 May 2006

Last Updated on STN: 10 Jun 2006

Entered Medline: 9 Jun 2006

ED Entered STN: 17 May 2006

Last Updated on STN: 10 Jun 2006

Entered Medline: 9 Jun 2006

AB BACKGROUND: Platelets participate in events that immediately precede acute myocardial infarction. Because platelets lack nuclear DNA but retain megakaryocyte-derived mRNAs, the platelet transcriptome provides a novel window on gene expression preceding acute coronary events. METHODS AND RESULTS: We profiled platelet mRNA from patients with acute ST-segment-elevation myocardial infarction (STEMI, n=16) or stable coronary artery disease (n=44). The platelet transcriptomes were analyzed and single-gene models constructed to identify candidate genes with differential expression. We validated 1 candidate gene product by performing a prospective, nested case-control study (n=255 case-control pairs) among apparently healthy women to assess the risk of future cardiovascular events (nonfatal myocardial infarction, nonfatal stroke, and cardiovascular death) associated with baseline plasma levels of the candidate protein. Platelets isolated from

STEMI and coronary artery disease patients contained 54 differentially expressed transcripts. The strongest discriminators of STEMI in the microarrays were CD69 (odds ratio 6.2, $P < 0.001$) and myeloid-related protein-14 (MRP-14; odds ratio 3.3, $P = 0.002$). Plasma levels of MRP-8/14 heterodimer were higher in STEMI patients (17.0 versus 8.0 microg/mL, $P < 0.001$). In the validation study, the risk of a first cardiovascular event increased with each increasing quartile of MRP- 8/14 (Ptrend <0.001) such that women with the highest levels had a 3.8-fold increase in risk of any vascular event ($P < 0.001$). Risks were independent of standard risk factors and C-reactive protein. CONCLUSIONS: The platelet transcriptome reveals quantitative differences between acute and stable coronary artery disease. MRP-14 expression increases before STEMI, and increasing plasma concentrations of MRP-8/14 among healthy individuals predict the risk of future cardiovascular events.

L7 ANSWER 25 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:964158 CAPLUS Full-text

DOCUMENT NUMBER: 146:118965

TITLE: S100 proteins in the pathogenesis of Kawasaki disease

AUTHOR(S): Burns, Jane C.

CORPORATE SOURCE: Division of Allergy, Immunology, and Rheumatology,
Department of Pediatrics, UCSD School of Medicine, La Jolla, CA, USA

SOURCE: Journal of the American College of Cardiology (2006),
48(6), 1265-1267

CODEN: JACCDI; ISSN: 0735-1097

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

ED Entered STN: 19 Sep 2006

AB A review. The research of Hirono et al. (2006) entitled "Expression of myeloid-related protein-8 and -14 in patients with acute Kawasaki disease" is reviewed with commentary and refs. The study of Hirono et al. found a markedly increased serum levels of myeloid-related protein-8 (MRP-8)/MRP14 in patients with Kawasaki disease (KD) compared with healthy controls. It provides support to the hypothesis that MRP8/MRpl4 heterodimers participate directly in the pathogenesis of the coronary artery vasculitis in KD and contributes to endothelial cell damage and transmigration of leukocytes into the arterial wall.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 26 OF 68 MEDLINE on STN DUPLICATE 12

ACCESSION NUMBER: 2006554271 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 16979015

TITLE: Expression of myeloid-related protein-8 and -14 in patients with acute Kawasaki disease.

AUTHOR: Hirono Keiich; Foell Dirk; Xing Yanlin; Miyagawa-Tomita Sachiko; Ye Fei; Ahlmann Martina; Vogl Thomas; Futatani Takeshi; Rui Chen; Yu Xianyi; Watanabe Kazuhiro; Wanatabe Sayaka; Tsubata Shinichi; Uese Keiichiro; Hashimoto Ikuo;

Ichida Fukiko; Nakazawa Makoto; Roth Johannes; Miyawaki Toshio

CORPORATE SOURCE: Department of Pediatrics, Toyama University, Toyama, Japan.

SOURCE: Journal of the American College of Cardiology, (2006 Sep 19)
Vol. 48, No. 6, pp. 1257-64. Electronic Publication: 2006-08-28.

Journal code: 8301365. E-ISSN: 1558-3597.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200610
ENTRY DATE: Entered STN: 19 Sep 2006
Last Updated on STN: 18 Oct 2006
Entered Medline: 17 Oct 2006

ED Entered STN: 19 Sep 2006
Last Updated on STN: 18 Oct 2006
Entered Medline: 17 Oct 2006

AB OBJECTIVES: This study investigated patients with acute Kawasaki disease (KD) to validate myeloid-related protein (MRP)-8/MRP-14 as a marker of disease activity and severity of coronary artery lesion development. BACKGROUND: Both MRP-8 and -14, which are S100-proteins secreted by activated neutrophils and monocytes, bind specifically to endothelial cells and induce thrombogenic and inflammatory responses in a variety of disease conditions. METHODS: We investigated 61 patients with acute KD and examined sequential changes in serum levels of MRP-8/MRP-14, messenger ribonucleic acid (mRNA) expression of MRP-8 and -14 in circulating granulocytes and monocytes, and amounts of MRP-8/MRP-14 bound to circulating endothelial cells. RESULTS: The serum MRP-8/MRP-14 levels as well as mRNA expressions of MRP-8 and -14 in granulocytes were strongly upregulated during the early stage of acute KD, and decreased dramatically within 24 h of intravenous immune globulin therapy ($p < 0.05$) in 45 responders. In contrast, in 16 nonresponders both of these increased after the initial treatment. The number of MRP-8/MRP-14-positive circulating endothelial cells was higher in patients with acute KD than in control patients and increased significantly by 2 weeks after the onset of KD, especially in patients in whom coronary artery lesions developed. CONCLUSIONS: We show for the first time that MRP-8/MRP-14 are exclusively secreted by granulocytes in patients with acute KD, and intravenous immune globulin treatment suppresses their gene expression. Serum levels of MRP-8/MRP-14 may be useful markers of disease activity, and the levels of MRP-8/MRP-14-positive circulating endothelial cell may predict the severity of vasculitis, confirming an important role for distinct inflammatory reactions in endothelium.

L7 ANSWER 27 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:994970 CAPLUS Full-text

DOCUMENT NUMBER: 145:452287

TITLE: Comparative proteomic analysis of mouse embryonic stem cells and neonatal-derived cardiomyocytes

AUTHOR(S): Baharvand, Hossein; Hajheidari, Mohsen; Zonouzi, Roseata; Ashtiani, Saeid Kazemi; Hosseinkhani, Saman; Salekdeh, Ghasem Hosseini

CORPORATE SOURCE: Department of Stem Cells, Royan Institute, Tehran, Iran

SOURCE: Biochemical and Biophysical Research Communications
(2006), 349(3), 1041-1049

CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 26 Sep 2006

AB Pluripotent embryonic stem cells (ESCs) spontaneously differentiate via embryo-like aggregates into cardiomyocytes. A thorough understanding of the mol. conditions in ESCs is necessary before other potential applications of these cells such as cell therapy can be materialized. We applied two dimensional electrophoresis to analyze and compare the proteome profiling of spontaneous mouse ESC-derived cardiomyocytes (ESC-DCs), undifferentiated mouse ESCs, and neonatal-derived cardiomyocytes (N-DCs). Ninety-five percent of the proteins detected on the ESC-DCs and N-DCs could be precisely paired with one other, whereas only twenty percent of the ESC proteins could be reliably matched with those on the ESC-DCs and N-DCs, suggesting a striking similarity between them. Having identified sixty proteins in the said three cell types,

we sought to provide possible explanations for their differential expression patterns and discuss their relevance to cell biol. This study provides a new insight into the gene expression pattern of differentiated cardiomyocytes and is further evidence for a close relation between ESC-DCs and N-DCs.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 28 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:779264 CAPLUS Full-text

DOCUMENT NUMBER: 146:220598

TITLE: Prophylactic aspirin therapy does not increase fecal calprotectin concentrations

AUTHOR(S): Montalto, Massimo; Curigliano, Valentina; Santoro, Luca; Lombardi, Mariaelena; Covino, Marcello; Cammarota, Giovanni; Dalvai, Sara; D'Onofrio, Ferruccio; Gasbarrini, Antonio; Gasbarrini, Giovanni

CORPORATE SOURCE: Departments of Internal Medicine bCardiology, Catholic University, Rome, Italy

SOURCE: European Journal of Gastroenterology & Hepatology (2006), 18(9), 965-967

CODEN: EJGHES; ISSN: 0954-691X

PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 08 Aug 2006

AB Non-steroidal anti-inflammatory drugs (NSAID) can induce enteropathy. Aspirin ingestion is associated with a lower small-intestinal inflammation than other NSAID. Faecal calprotectin concns. have recently been proposed as a simple non-invasive test to identify NSAID enteropathy. The aim of our pilot study was to evaluate calprotectin concns. in patients on treatment with low-dose aspirin. Twenty-two patients on prophylactic treatment with aspirin were recruited. Twenty-five healthy volunteers were enrolled as a control group. Faecal calprotectin concns. were detd. by ELISA. Statistical anal. was performed by t-test for unpaired data. The mean fecal calprotectin concentration in patients ($57.95 \pm 44.28 \mu\text{g/g}$) did not show significant differences compared with controls ($45.76 \pm 26.45 \mu\text{g/g}$; $P = 0.251$). We found that low-dose aspirin does not induce an increase in fecal calprotectin increase.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 29 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2007:124739 BIOSIS Full-text

DOCUMENT NUMBER: PREV200700123958

TITLE: The presence of congestive heart failure modulates the ability of ejection fraction to predict mortality in patients admitted for possible myocardial infarction.

AUTHOR(S): Kontos, Michael C. [Reprint Author]; Khasawinah, Tariq; Jamal, Sameer M.; Garg, Rajat; Roberts, Charlotte S.; Ornato, Joseph P.; Tatum, James L.; Jesse, Robert L.

CORPORATE SOURCE: Virginia Commonwealth Univ, Richmond, VA USA

SOURCE: Circulation, (OCT 31 2006) Vol. 114, No. 18, Suppl. S, pp. 744. Meeting Info.: 79th Annual Scientific Session of the American-Heart-Association. Chicago, IL, USA. November 12 -15, 2006. Amer Heart Assoc.

CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

ED Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

AB Background: Both congestive heart failure (CHF) and ejection fraction (EF) have been shown to be powerful predictors of outcome in ACS patients (pts). However, few studies have included both variables, as EF has been measured only infrequently. Methods: Consecutive pts admitted for MI exclusion underwent serial assessment of CK, CK-MB and Tnl. CHF was assessed at the time of admission using physical exam and chest x-ray findings. Abnormal EF was defined as $\leq 40\%$. One year mortality was assessed and results reported based on the presence of abnormal EF, Tnl elevations and CHF. Results: Over a 4 year period, 4,343 consecutive pts without ST elevation on the initial ECG were admitted, of whom 3,584 (83%, including 94% of those who were Tnl (+)) had EF assessed. A total of 652 pts (18%) had Tnl elevations. One year mortality is shown in the Figure. Pts with EF $\leq 40\%$ had a significantly higher ($p < 0.001$) mortality compared to those with an EF $> 40\%$, both in the presence and absence of CHF. For each EF category, the presence of CHF significantly increased mortality two-fold or more ($p < 0.001$) in all groups. Abnormal EF or CHF resulted in an approximately two-fold increase in mortality in pts who did and did not have Tnl elevations. Conclusions: The presence of CHF in pts with both normal and abnormal EF doubles mortality in pts admitted for possible MI. [GRAPHICS] The 4th day (1025 ± 123 ng/ml). The peak MRP 8/14 levels were positively correlated with peak CRP levels ($r=0.67$, $p < 0.001$) and peak CK levels ($r=0.55$, $p < 0.01$). In patients with major early phase complications such as cardiac or papillary muscle rupture, and severe heart failure ($n=8$), the serum MRP 8/14 levels on the 1st hospital day were significantly higher than in those without complications ($n=17$) (1033 ± 173 ng/ml vs. 555 ± 51 ng/ml, $p < 0.001$). Immunohistochemically, MRP 8/14 was specifically and strongly positive in the cytoplasm of neutrophils and macrophages in the necrotic myocardium from AMI patients. Conclusions: These findings imply that MRP 8/14, produced by neutrophils and macrophages, plays an important role in the pathogenesis of AMI. The increase in serum MRP 8/14 levels may predict the severity and risk of early phase complications in AMI.

L7 ANSWER 30 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2007:124740 BIOSIS Full-text

DOCUMENT NUMBER: PREV200700123959

TITLE: Decreased serum levels of interferon gamma inducible protein 10 (IP-10) in acute myocardial infarction patients after successful primary percutaneous coronary intervention are associated with a smaller infarct size.

AUTHOR(S): Hirohata, Satoshi [Reprint Author]; Koten, Kazuya; Usui, Shinichi; Ogawa, Hiroko; Yamawaki, Hitoshi; Obika, Masanari; Iwamoto, Mutsurni; Shiratori, Yasushi; Kusachi, Shozo; Ohe, Tohru

CORPORATE SOURCE: Okayama Univ, Grad Sch Med Dent and Pharmaceut Sci, Okayama 7008530, Japan

SOURCE: Circulation, (OCT 31 2006) Vol. 114, No. 18, Suppl. S, pp. 744. Meeting Info.: 79th Annual Scientific Session of the American Heart-Association. Chicago, IL, USA. November 12 -15, 2006. Amer Heart Assoc. CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

ED Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

AB Background: Interferon gamma inducible protein 10 (IP-10) is a CXC chemokine that is transiently induced in ischemia/reperfusion and an appropriate decrease of induced IP-10 in the reperfused heart is important for infarct healing. However, the serum IP-10 level in myocardial infarction (MI) patients with percutaneous coronary intervention (PCI) and its changes with

time have never been reported. Methods: We prospectively studied 23 consecutive patients with first MI who received successful primary PCI within 24 hours. Blood samples were collected after the primary PCI and on days 3, 7 and 28. Changes of the serum IP-10 level between the day of admission and 3 d (delta 3 d IP-10) were examined. Results: Serum IP-10 level in MI patients changed compared with normal levels and its alteration was statistically significant. The mean value of delta 3 d IP-10 in the better EF group (> 56.9%) was -9.0 pg/ml, while it was 28.4 pg/ml in the worse EF group. Delta 3 d IP-10 was significantly correlated with infarct size, as indicated by max creatine kinase (CK). Stepwise multiple regression analysis revealed that delta 3 d IP-10 was an independent predictor for max CK. Conclusions: These data suggest that decreased serum IP-10 level within 3 days correlate with a smaller infarct size in patients with acute MI who undergo successful primary PCI. [GRAPHICS]sible MI. [GRAPHICS]he 4th day (1025 +/- 123 ng/ml). The peak MRP 8/14 levels were positively correlated with peak CRP levels ($r=0.67$, $p < 0.001$) and peak CK levels ($r=0.55$, $p < 0.01$). In patients with major early phase complications such as cardiac or papillary muscle rupture, and severe heart failure ($n=8$), the serum MRP 8/14 levels on the 1st hospital day were significantly higher than in those without complications ($n=17$) (1033 +/- 173 ng/ml vs. 555 +/- 51 ng/ml, $p < 0.001$). Immunohistochemically, MRP 8/14 was specifically and strongly positive in the cytoplasm of neutrophils and macrophages in the necrotic myocardium from AMI patients. Conclusions: These findings imply that MRP 8/14, produced by neutrophils and macrophages, plays an important role in the pathogenesis of AMI. The increase in serum MRP 8/14 levels may predict the severity and risk of early phase complications in AMI.

L7 ANSWER 31 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2007:124738 BIOSIS [Full-text](#)

DOCUMENT NUMBER: PREV200700123957

TITLE: Enhanced expression of myeloid-related protein complex (MRP 8/14) predicts the severity and risk of complications in acute myocardial infarction.

AUTHOR(S): Katashima, Takashi [Reprint Author]; Terasaki, Fumio; Shimomura, Hiroaki; Otsuka, Kaoru; Tsukada, Bin; Murakami, Shougo; Kitaura, Yasushi; Ikemoto, Masaki; Fujita, Masatoshi

CORPORATE SOURCE: Osaka Med Coll, Takatsuki, Osaka, Japan

SOURCE: Circulation, (OCT 31 2006) Vol. 114, No. 18, Suppl. S, pp. 743-744.

Meeting Info.: 79th Annual Scientific Session of the American-Heart-Association. Chicago, IL, USA. November 12 -15, 2006. Amer Heart Assoc.

CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

ED Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

AB Introduction: Myeloid-related protein complex (MRP 8/14), a hetero dimer of MRP 8 and 14, is expressed in activated human neutrophils and macrophages. Although MRP 8/14 is reported to be involved in the inflammatory process of various diseases, its expression in acute myocardial infarction (AMI) has not been clarified. Hypothesis: We assessed the hypothesis that serum levels of MRP 8/14 are increased in patients with AMI, and the expression of MRP 8/14 is upregulated in the myocardium of AMI patients. Methods: Serum MRP 8/14 levels were measured using a sandwich enzyme-linked immunosorbent assay system in 25 consecutive patients [68 +/- 2 (SE) years, 17 men and 8 women] with AMI during the acute period continuously, from the onset to the 10th hospital day. Sera from 30 normal volunteers served as controls. All AMI patients underwent

successful primary percutaneous coronary intervention within 12 hours after the onset of symptoms. In AMI patients, serum levels of C-reactive protein (CRP) and creatine kinase (CK) were also measured. In addition, we immunohistochemically examined the expression profiles of MRP 8/14 in the autopsied myocardium from 4 patients with AMI. Results: In AMI patients, serum MRP 8/14 levels on the 1st day were significantly higher than in normal controls [636 +/- 86 ng/ml vs. 230 +/- 21 ng/ml, $p < 0.0001$] and reached the peak levels on the 4th day (1025 +/- 123 ng/ml). The peak MRP 8/14 levels were positively correlated with peak CRP levels ($r=0.67$, $p < 0.001$) and peak CK levels ($r=0.55$, $p < 0.01$). In patients with major early phase complications such as cardiac or papillary muscle rupture, and severe heart failure ($n=8$), the serum MRP 8/14 levels on the 1st hospital day were significantly higher than in those without complications ($n=17$) (1033 +/- 173 ng/ml vs. 555 +/- 51 ng/ml, $p < 0.001$). Immunohistochemically, MRP 8/14 was specifically and strongly positive in the cytoplasm of neutrophils and macrophages in the necrotic myocardium from AMI patients. Conclusions: These findings imply that MRP 8/14, produced by neutrophils and macrophages, plays an important role in the pathogenesis of AMI. The increase in serum MRP 8/14 levels may predict the severity and risk of early phase complications in AMI.

L7 ANSWER 32 OF 68 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006430740 EMBASE Full-text

TITLE: Receptor for Advanced Glycation End Products and the Cardiovascular Complications of Diabetes and Beyond: Lessons from AGEing.

AUTHOR: Yan, Shi Fang; Yan, Shi Du; Herold, Kevan; Ramsamy, Ravichandran; Schmidt, Ann Marie, Dr. (correspondence)

CORPORATE SOURCE: Columbia University Medical Center, 630 West 168th St, P and S 17-501, New York, NY 10032, United States. ams11@columbia.edu

SOURCE: Endocrinology and Metabolism Clinics of North America, (Sep 2006) Vol. 35, No. 3, pp. 511-524.

Refs: 70

ISSN: 0889-8529 CODEN: ECNAER

PUBLISHER IDENT.: S 0889-8529(06)00043-0

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
003 Endocrinology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
006 Internal Medicine

LANGUAGE: English

ENTRY DATE: Entered STN: 28 Sep 2006

Last Updated on STN: 28 Sep 2006

ED Entered STN: 28 Sep 2006

Last Updated on STN: 28 Sep 2006

AB Although the RAGE axis and its blockade have yet to be tested in clinical trials, studies linking RAGE expression and levels of plasma sRAGE to human diabetes and cardiovascular disease suggest that this axis is probably relevant to the complications of hyperglycemia and inflammation. In perturbed vasculature, stresses induced by stimuli such as hyperlipidemia, physical injury, infection, or hyperglycemia may lead to upregulation of RAGE, thereby stimulating endothelial upregulation of inflammatory molecules and the recruitment of S100/calgranulin and HMGB1-bearing activated inflammatory cells. S100/calgranulins and HMGB1 may be released by these inflammatory cells in the vasculature. Thus, inflammatory cells bearing S100/calgranulins and HMGB1 may contribute integrally to mechanisms that initiate and advance atherosclerosis and vascular perturbation. In cardiovascular disease, particularly that consequent to long-term diabetes, multiple hits contribute

to the initiation and progression of atherosclerosis, and, in advanced stages, to ischemia-reperfusion events. Efforts to delineate the precise steps at which RAGE plays contributory roles are critical in the optimal design of clinical trials to test these concepts. .COPYRGT. 2006 Elsevier Inc. All rights reserved.

L7 ANSWER 33 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:123704 BIOSIS Full-text
DOCUMENT NUMBER: PREV200700122923
TITLE: Dynamic change and pivotal role of myeloid-related protein
(MRP) 8 and MRP14 in acute kawasaki disease.
AUTHOR(S): Hirono, Keitchi [Reprint Author]; Foell, Dirk; Xing, Yanling;
Miyagawa-Tomita, Sachiko; Ye, Fei; Watanabe, Kazuhiro; Watanabe, Sayaka; Uese,
Kelichirou; Nakazawa, Makoto; Ichida, Fukiko; Roth, Johannes; Miyawaki, Toshio
CORPORATE SOURCE: Toyama Univ, Fac Med, Dept Pediat, Toyama 930, Japan
SOURCE: Circulation, (OCT 31 2006) Vol. 114, No. 18, Suppl. S, pp. 503.
Meeting Info.: 79th Annual Scientific Session of the American-
Heart-Association. Chicago, IL, USA. November 12 -15, 2006. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Feb 2007
Last Updated on STN: 22 Feb 2007

ED Entered STN: 22 Feb 2007

Last Updated on STN: 22 Feb 2007

AB Background: Myeloid-related protein (MRP) 8 and MRP14, which are S100-proteins secreted by activated neutrophils and monocytes, bind specifically to endothelial cells, and induce thrombogenic and inflammatory responses in a variety of disease conditions. Objective: The aim of our study was to investigate the role of MRP8/MRP14 in acute Kawasaki disease (KD) and to validate MRP8/MRP14 as a marker of disease activity and severity of coronary artery lesion (CAL) development. Methods: We investigated 61 patients with acute KD differentiated by response to intravenous immune globulin (IVIG) treatment, and examined sequential changes in serum levels of MRP8/MRP14, mRNA expression of MRP8 and MRP14, intracellular expression of MRP8/MRP14 protein, and amounts of MRP8/MRP14 bound to circulating endothelial cells. Results: The initial serum levels of MRP8/MRP14 were elevated in all patients with KD and decreased within 24 hours of IVIG ($P<0.05$) in the group of 45 responders. In contrast, in 16 non-responders the serum MRP8/MRP14 levels increased after the initial treatment. mRNA expression of MRP8 and MRP14 were significantly higher in the granulocytes of acute KD responders prior to treatment with IVIG, decreasing significantly within 24 hours of IVIG treatment ($P<0.05$). The both of expression in non-responders increased after the initial treatment. Intracellular expression of MRP8/MRP14 protein in neutrophils of acute KD patients are markedly higher than that of normal controls, and gradually increased after IVIG treatment in all patients. The number of MRP8/MRP14-positive circulating endothelial cells was higher in patients with acute KD than controls and this increased significantly by 2 weeks after the onset of KD, especially in patients who developed CAL. Conclusions: We show for the first time that MRP8/MRP14 are exclusively expressed and rapidly secreted by granulocytes in patients with acute KD, and successful IVIG treatment suppresses their gene expression. Serum levels of MRP8/MRP14 may be useful markers of disease activity, and the levels of MRP8/MRP14 positive circulating endothelial cell predict the severity of vasculitis, confirming an important role for distinct inflammatory reactions in endothelium.

L7 ANSWER 34 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:653812 CAPLUS Full-text
DOCUMENT NUMBER: 145:436019
TITLE: Inhibition of LINE-1 expression in the heart decreases
ischemic damage by activation of Akt/PKB signaling
AUTHOR(S): Lucchinetti, Eliana; Feng, Jianhua; da Silva, Rafaela;
Tolstonog, Genrich V.; Schaub, Marcus C.; Schumann, Gerald G.; Zaugg, Michael
CORPORATE SOURCE: Cardiovascular Anesthesia Research Laboratory, Institute
of Anesthesiology, University Hospital Zurich, Zurich, Switz.
SOURCE: Physiological Genomics (2006), 25(2), 314-324
CODEN: PHGEFP; ISSN: 1094-8341
URL:

<http://physiolgenomics.physiology.org/cgi/reprint/25/2/314>

PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English

ED Entered STN: 06 Jul 2006

AB Microarray analyses indicate that ischemic and pharmacol. preconditioning suppress overexpression of the non-long terminal repeat retrotransposon long interspersed nuclear element 1 (LINE-1, L1) after ischemia-reperfusion in the rat heart. We tested whether L1 overexpression is mechanistically involved in postischemic myocardial damage. Isolated, perfused rat hearts were treated with antisense or scrambled oligonucleotides (ODNs) against L1 for 60 min and exposed to 40 min of ischemia followed by 60 min of reperfusion. Functional recovery and infarct size were measured. Effective nuclear uptake was determined by FITC-labeled ODNs, and downregulation of L1 transcription was confirmed by RT-PCR. Immunoblot anal. was used to assess changes in expression levels of the L1-encoded proteins ORF1p and ORF2p. Immunohistochem. was performed to localize ORF1/2 proteins in cardiac tissue. Effects of ODNs on prosurvival protein kinase B (Akt/PKB) expression and activity were also determined. Antisense ODNs against L1 prevented L1 burst after ischemia-reperfusion. Inhibition of L1 increased Akt/PKB β expression, enhanced phosphorylation of PKB at serine 473, and markedly improved postischemic functional recovery and decreased infarct size. Antisense ODN-mediated protection was abolished by LY-294002, confirming the involvement of the Akt/PKB survival pathway. ORF1p and ORF2p were found to be expressed in rat heart. ORF1p showed a predominantly nuclear localization in cardiomyocytes, whereas ORF2p was exclusively present in endothelial cells. ORF1p levels increased in response to ischemia, which was reversed by antisense ODN treatment. No significant changes in ORF2p were noted. Our results demonstrate that L1 suppression favorably affects postischemic outcome in the heart. Modifying transcriptional activity of L1 may represent a novel anti-ischemic therapeutic strategy.

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 35 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2007:47897 BIOSIS Full-text
DOCUMENT NUMBER: PREV200700048758
TITLE: Early S100A8/A9 protein elevation in patients with ACS.
AUTHOR(S): Eggers, K. [Reprint Author]; Lorenz, M.; Laule, M.; Remppis, A.; Baumann, G.; Stangl, K.; Stangl, V.
CORPORATE SOURCE: Univ Heidelberg, Med Klin 3, Heidelberg, Germany
SOURCE: European Heart Journal, (AUG 2006) Vol. 27, No. Suppl. 1, pp. 217.

Meeting Info.: World Congress of Cardiology. Barcelona, SPAIN. September 02 -06, 2006.

CODEN: EHJODF. ISSN: 0195-668X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English

ENTRY DATE: Entered STN: 10 Jan 2007
Last Updated on STN: 10 Jan 2007
ED Entered STN: 10 Jan 2007
Last Updated on STN: 10 Jan 2007

L7 ANSWER 36 OF 68 MEDLINE on STN DUPLICATE 13
ACCESSION NUMBER: 2006420573 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16845180
TITLE: Acute coronary artery injury in dogs following administration
of CI-1034, an endothelin A receptor antagonist.
AUTHOR: McDuffie J Eric; Yu Xinwen; Sobocinski Gregg; Song Yunling;
Chupka Jonathan; Albassam Mudher
CORPORATE SOURCE: Esperion Therapeutics, a Division of Pfizer Global Research and
Development, Drug Safety Evaluation, Plymouth, MI 48170, USA..
eric.mcduffie@pfizer.com
SOURCE: Cardiovascular toxicology, (2006) Vol. 6, No. 1, pp. 25-38.
Journal code: 101135818. ISSN: 1530-7905.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200608
ENTRY DATE: Entered STN: 18 Jul 2006
Last Updated on STN: 19 Aug 2006
Entered Medline: 18 Aug 2006

ED Entered STN: 18 Jul 2006
Last Updated on STN: 19 Aug 2006
Entered Medline: 18 Aug 2006

AB The objective of this study was to characterize acute coronary artery injury
evoked by the endothelin A receptor (ETAR) antagonist, CI-1034. Male dogs (n
= 5) were intravenously administered CI-1034 at 120 mg/kg for 4 d. Control
animals (n = 3) received vehicle. Macroscopically, drug-related hemorrhage
was observed in the right coronary groove and atrium. Histologically,
drugrelated coronary changes were characterized as medial hemorrhage and
necrosis, with mixed inflammatory-cell infiltrates in the adventitia and
media. Immunohistochemistry staining indicated increased expression of
inducible nitric oxide synthase (iNOS), cleaved caspase-3, and S100A8/A9
(within in monocytes and neutrophils) proteins in coronary arteries of CI-
1034-treated animals. However, there were similar expression levels of
endothelial nitric oxide synthase (eNOS) among control and CI-1034-treated
animals. Significant drug-related nitric oxide (NO) accumulation occurred on
days 1 through 4 in serum. Increased interleukin (IL)-6 and fibrinogen in
plasma and serum amyloid A (SAA) occurred on days 2 through 5 in CI-1034-
treated animals. Increased levels of NO accumulation in serum; increased IL-6
and fibrinogen levels in plasma; increased SAA levels; and increased
expressions of iNOS, cleaved caspase-3, and S100A8/A9 complex appear to be
characteristic of CI-1034-induced acute vascular injury in dogs.

L7 ANSWER 37 OF 68 MEDLINE on STN
ACCESSION NUMBER: 2006351364 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16573830
TITLE: Increased proinflammatory endothelial response to S100A8/A9
after preactivation through advanced glycation end products.
AUTHOR: Ehlermann Philipp; Eggers Kai; Bierhaus Angelika; Most Patrick;
Weichenhan Dieter; Greten Johannes; Nawroth Peter P; Katus Hugo A; Remppis Andrew
CORPORATE SOURCE: Universitat Heidelberg, Abteilung Innere Medizin III,
Heidelberg, Germany.. philipp.ehlermann@med.uni-heidelberg.de
SOURCE: Cardiovascular diabetology, (2006) Vol. 5, pp. 6. Electronic
Publication: 2006-03-30.

Journal code: 101147637. E-ISSN: 1475-2840.
PUB. COUNTRY: England; United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200606
ENTRY DATE: Entered STN: 13 Jun 2006
Last Updated on STN: 24 Jun 2006
Entered Medline: 23 Jun 2006

ED Entered STN: 13 Jun 2006
Last Updated on STN: 24 Jun 2006
Entered Medline: 23 Jun 2006

AB BACKGROUND: Atherosclerosis is an inflammatory disease in which a perpetuated activation of NFkappaB via the RAGE (receptor for advanced glycation end products)-MAPK signalling pathway may play an important pathogenetic role. As recently S100 proteins have been identified as ligands of RAGE, we sought to determine the effects of the proinflammatory heterodimer of S100A8/S100A9 on the RAGE-NFkappaB mediated induction of proinflammatory gene expression. METHODS: Human umbilical vein endothelial cells (HUVEC) were preincubated for 72 h with AGE-albumin or unmodified albumin for control, whereas AGE-albumin induction resulted in an upregulation of RAGE. Following this preactivation, cells were stimulated for 48 h with heterodimeric human recombinant S100A8/S100A9. RESULTS: Heterodimeric S100A8/S100A9 enhanced secretion of IL-6, ICAM-1, VCAM-1 and MCP1 in AGE-albumin pretreated HUVEC in a dose dependent manner. These effects could not be detected after stimulation with the homodimeric proteins S100A8, S100A9, S100A1 and S100B. The effects of heterodimeric S100A8/S100A9 were reduced by inhibition of the MAP-kinase pathways ERK1/2 and p38 by PD 98059 and SB 203580, respectively. CONCLUSION: The heterodimeric S100A8/S100A9 might therefore play a hitherto unknown role in triggering atherosclerosis in diabetes and renal failure, pathophysiological entities associated with a high AGE burden. Thus, blocking heterodimeric S100A8/S100A9 might represent a novel therapeutic modality in treating atherosclerosis.

L7 ANSWER 38 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 14

ACCESSION NUMBER: 2006:466659 CAPLUS Full-text
DOCUMENT NUMBER: 145:268327

TITLE: Increased proinflammatory endothelial response to S100A8/A9 after preactivation through advanced glycation end products
AUTHOR(S): Ehlermann, Philipp; Eggers, Kai; Bierhaus, Angelika; Most, Patrick; Weichenhan, Dieter; Greten, Johannes; Nawroth, Peter P.; Katus, Hugo A.; Remppis, Andrew
CORPORATE SOURCE: Abteilung Innere Medizin III, Universitaet Heidelberg, Heidelberg, Germany
SOURCE: Cardiovascular Diabetology (2006), 5, No pp. given
CODEN: CDAIAZ; ISSN: 1475-2840
URL: <http://www.cardiab.com/content/pdf/1475-2840-5-6.pdf>

PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English

ED Entered STN: 18 May 2006

AB Background: Atherosclerosis is an inflammatory disease in which a perpetuated activation of NFkappaB via the RAGE (receptor for advanced glycation end products)-MAPK signaling pathway may play an important pathogenetic role. As recently S100 proteins have been identified as ligands of RAGE, we sought to determine the effects of the proinflammatory heterodimer of S100A8/S100A9 on the RAGE-NFkappaB mediated induction of proinflammatory gene expression. Methods: Human umbilical vein endothelial cells (HUVEC) were preincubated for

72 h with AGE-albumin or unmodified albumin for control, whereas AGE-albumin induction resulted in an upregulation of RAGE. Following this preactivation, cells were stimulated for 48 h with heterodimeric human recombinant S100A8/S100A9. Results: Heterodimeric S100A8/S100A9 enhanced secretion of IL-6, ICAM-1, VCAM-1 and MCP1 in AGE-albumin pretreated HUVEC in a dose dependent manner. These effects could not be detected after stimulation with the homodimeric proteins S100A8, S100A9, S100A1 and S100B. The effects of heterodimeric S100A8/S100A9 were reduced by inhibition of the MAP-kinase pathways ERK1/2 and p38 by PD 98059 and SB 203580, resp. Conclusions: The heterodimeric S100A8/S100A9 might therefore play a hitherto unknown role in triggering atherosclerosis in diabetes and renal failure, pathophysiol. entities associated with a high AGE burden. Thus, blocking heterodimeric S100A8/S100A9 might represent a novel therapeutic modality in treating atherosclerosis.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 39 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2005:141350 CAPLUS Full-text

DOCUMENT NUMBER: 142:216622

TITLE: Secreted polypeptide species reduced in cardiovascular disorders, and motilin expression related to intestinal motility disorders

INVENTOR(S): Argoud-Puy, Guilaine; Bederr, Nassima; Bougueleret, Lydie; Cusin, Isabelle; Mahe, Eva; Niknejad, Anne; Reffas, Samia; Rose, Keith; Saudrais, Cedric; Scherer, Andreas; Papoian, Ruben

PATENT ASSIGNEE(S): Genova Ltd., Bermuda; Novartis A.-G.; Novartis Pharma G.m.b.H.

SOURCE: PCT Int. Appl., 349 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005015206	A2	20050217	WO 2004-EP8860	20040806
WO 2005015206	A3	20050609		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1654545	A2	20060510	EP 2004-763890	20040806
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007501605	T	20070201	JP 2006-522332	20040806
PRIORITY APPLN. INFO.:				
			US 2003-493599P	P 20030808
			US 2003-493836P	P 20030808
			US 2003-493867P	P 20030808
			US 2003-493985P	P 20030808
			WO 2004-EP8860	W 20040806

ED Entered STN: 18 Feb 2005

AB The invention discloses human secreted polypeptides that circulate at a decreased level in the plasma of patients with cardiovascular disorders. Thus, 254 Cardiovascular disorder Plasma Polypeptides (CPP 149-402) are

identified, by reverse-phase HPLC and mass spectrometry of tryptic peptides, that are differentially reduced in concentration in plasma from individuals with coronary artery disease compared to control plasma. Over-expression of CPP 232, identified as motilin (SwissProt accession number P12872), is also shown to affect the expression of a number of genes/proteins related to intestinal motility, apoptosis pathway, proteosome and ubiquitin pathways, and rRNAs and proteins. Thus, expression of CPP 232 or the genes it regulated may be used for treatment, diagnosis, and monitoring of biliary cirrhosis, gallstones, celiac disease, and other intestinal motility disorders. The invention also provides methods of using compns. including the polypeptides, polynucleotides encoding them, and antibodies specific for these polypeptides, for diagnosis, prognosis, and for drug development.

L7 ANSWER 40 OF 68 MEDLINE on STN DUPLICATE 15
 ACCESSION NUMBER: 2005657019 MEDLINE Full-text
 DOCUMENT NUMBER: PubMed ID: 16216873
 TITLE: S100A8 and S100A9 in human arterial wall. Implications for atherogenesis.
 AUTHOR: McCormick Michelle M; Rahimi Farid; Bobryshev Yuri V; Gaus Katharina; Zreiqat Hala; Cai Hong; Lord Reginald S A; Geczy Carolyn L
 CORPORATE SOURCE: School of Medical Sciences, St. Vincent's Hospital, University of New South Wales, Sydney, Australia.
 SOURCE: The Journal of biological chemistry, (2005 Dec 16) Vol. 280, No. 50, pp. 41521-9. Electronic Publication: 2005-10-10.
 Journal code: 2985121R. ISSN: 0021-9258.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200602
 ENTRY DATE: Entered STN: 18 Dec 2005
 Last Updated on STN: 8 Feb 2006
 Entered Medline: 7 Feb 2006
 ED Entered STN: 18 Dec 2005
 Last Updated on STN: 8 Feb 2006
 Entered Medline: 7 Feb 2006
 AB Atherogenesis is a complex process involving inflammation. S100A8 and S100A9, the Ca²⁺-binding neutrophil cytosolic proteins, are associated with innate immunity and regulate processes leading to leukocyte adhesion and transmigration. In neutrophils and monocytes the S100A8-S100A9 complex regulates phosphorylation, NADPH-oxidase activity, and fatty acid transport. The proteins have anti-microbial properties, and S100A8 may play a role in oxidant defense in inflammation. Murine S100A8 is regulated by inflammatory mediators and recruits macrophages with a proatherogenic phenotype. S100A9 but not S100A8 was found in macrophages in ApoE^{-/-} murine atherosclerotic lesions, whereas both proteins are expressed in human giant cell arteritis. Here we demonstrate S100A8 and S100A9 protein and mRNA in macrophages, foam cells, and neovessels in human atheroma. Monomeric and complexed forms were detected in plaque extracts. S100A9 was strongly expressed in calcifying areas and the surrounding extracellular matrix. Vascular matrix vesicles contain high levels of Ca²⁺-binding proteins and phospholipids that regulate calcification. Matrix vesicles characterized by electron microscopy, x-ray microanalysis, nucleoside triphosphate pyrophosphohydrolase assay and cholesterol/phospholipid analysis contained predominantly S100A9. We propose that S100A9 associated with lipid structures in matrix vesicles may influence phospholipid-Ca²⁺ binding properties to promote dystrophic calcification. S100A8 and S100A9 were more sensitive to hypochlorite oxidation than albumin or low density lipoprotein and immunoaffinity confirmed S100A8-S100A9

complexes; some were resistant to reduction, suggesting that hypochlorite may contribute to protein cross-linking. S100A8 and S100A9 in atherosclerotic plaque and calcifying matrix vesicles may significantly influence redox- and Ca2+-dependent processes during atherogenesis and its chronic complications, particularly dystrophic calcification.

L7 ANSWER 41 OF 68 MEDLINE on STN DUPLICATE 16
ACCESSION NUMBER: 2005209106 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 15843588
TITLE: Gene expression profiling of the effect of high-dose intravenous Ig in patients with Kawasaki disease.
AUTHOR: Abe Jun; Jibiki Toshiaki; Noma Seiji; Nakajima Toshiharu; Saito Hirohisa; Terai Masaru
CORPORATE SOURCE: Department of Allergy and Immunology, National Research Institute for Child Health and Development, Tokyo, Japan.. jabe@nch.go.jp
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2005 May 1) Vol. 174, No. 9, pp. 5837-45.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200506
ENTRY DATE: Entered STN: 22 Apr 2005
Last Updated on STN: 22 Jun 2005
Entered Medline: 21 Jun 2005
ED Entered STN: 22 Apr 2005
Last Updated on STN: 22 Jun 2005
Entered Medline: 21 Jun 2005
AB Kawasaki disease (KD) is an acute vasculitis of infants and young children, preferentially affecting the coronary arteries. Intravenous infusion of high dose Ig (IVIG) effectively reduces systemic inflammation and prevents coronary artery lesions in KD. To investigate the mechanisms underlying the therapeutic effects of IVIG, we examined gene expression profiles of PBMC and purified monocytes obtained from acute patients before and after IVIG therapy. The results suggest that IVIG suppresses activated monocytes and macrophages by altering various functional aspects of the genes of KD patients. Among the 18 commonly decreased transcripts in both PBMC and purified monocytes, we selected six genes, FCGR1A, FCGR3A, CCR2, ADM, S100A9, and S100A12, and confirmed the microarray results by real-time RT-PCR. Moreover, the expressions of FcgammaRI and FcgammaRIII on monocytes were reduced after IVIG. Plasma S100A8/A9 heterocomplex, but not S100A9, levels were elevated in patients with acute KD compared with those in febrile controls. Furthermore, S100A8/A9 was rapidly down-regulated in response to IVIG therapy. Persistent elevation of S100A8/A9 after IVIG was found in patients who later developed coronary aneurysms. These results indicate that the effects of IVIG in KD may be mediated by suppression of an array of immune activation genes in monocytes, including those activating FcgammaRs and the S100A8/A9 heterocomplex.

L7 ANSWER 42 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:59624 BIOSIS Full-text
DOCUMENT NUMBER: PREV200600047901
TITLE: Enhanced expression of myeloid related protein complex (MRP 8/14) in multinucleated giant cells and macrophages in granulomas of patients with active cardiac sarcoidosis.
AUTHOR(S): Terasaki, Fumio [Reprint Author]; Shimomura, Hiroaki; Tsukada,

Bin; Otsuka, Koji; Kitaura, Yasushi; Ikemoto, Masaki; Fujija, Masatoshi
CORPORATE SOURCE: Osaka Med Coll, Takatsuki, Osaka 569, Japan
SOURCE: Circulation, (OCT 25 2005) Vol. 112, No. 17, Suppl. S, pp.
U468.

Meeting Info.: 78th Annual Scientific Session of the American-
Heart-Association. Dallas, TX, USA. November 13 -16, 2005. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 4 Jan 2006

Last Updated on STN: 4 Jan 2006

ED Entered STN: 4 Jan 2006

Last Updated on STN: 4 Jan 2006

L7 ANSWER 43 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2005:1112606 CAPLUS Full-text

DOCUMENT NUMBER: 144:168189

TITLE: The RAGE Axis and Endothelial Dysfunction: Maladaptive
Roles in the Diabetic Vasculature and Beyond

AUTHOR(S): Ramasamy, Ravichandran; Yan, Shi Fang; Schmidt, Ann Marie

CORPORATE SOURCE: Division of Surgical Science, Department of Surgery,
Columbia University Medical Center, New York, NY, USA

SOURCE: Trends in Cardiovascular Medicine (2005), 15(7), 237-243
CODEN: TCMDEQ; ISSN: 1050-1738

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

ED Entered STN: 17 Oct 2005

AB A review. Receptor for advanced glycation end product (RAGE) is a member of
the Ig superfamily of cell surface mols. The ligand-RAGE axis is emerging as
a central mechanism linked to vascular injury and atherosclerosis in diabetes
and in euglycemia. The repertoire of RAGE ligands, including advanced
glycation end products, S100/calgranulins, high-mobility group box 1, amyloid-
 β peptide, and Mac-1, transcends RAGE biol. from specifically the science of
diabetic complications to central aspects of the inflammatory response and
oxidative stress. Expts. in cell culture and in vivo support the notion that
interaction of RAGE ligands with RAGE activates key signal transduction
pathways that modulate fundamental cellular properties, thereby leading to
vascular and inflammatory cell perturbation. These considerations support the
premise that the ligand-RAGE axis may be an important target for therapeutic
intervention in cardiovascular disease and, fundamentally, in initiation and
amplification of inflammatory responses.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 44 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2005:1081525 CAPLUS Full-text

DOCUMENT NUMBER: 145:109976

TITLE: Gene expression changes in leukocytes during
cardiopulmonary bypass are dependent on circuit coating

AUTHOR(S): Seeburger, Joerg; Hoffmann, Jan; Wendel, Hans Peter;
Ziemer, Gerhard; Aebert, Hermann

CORPORATE SOURCE: Department of Thoracic, Cardiac and Vascular Surgery,
Eberhard Karls University, Tuebingen, Germany

SOURCE: Circulation (2005), 112(9, Suppl.), I/224-I/228
CODEN: CIRCAZ; ISSN: 0009-7322

PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 10 Oct 2005

AB Background-Cardiopulmonary bypass (CPB) results in a systemic inflammatory response. Leukocytes play a crucial role in inflammatory reactions. Their gene expression profile in the context of CPB is unknown. Methods and Results-In a prospective, randomized, and double-blind clin. trial, 12 male patients underwent elective coronary artery bypass grafting with either heparin-coated (group H) or protein-coated (group P) CPB circuits. Oligonucleotide microarray analyses of 22 283 genes were performed on circulating leukocytes, collected immediately before surgery and 6 h after CPB. Microarray results were validated with real-time polymerase chain reaction. All patients had uneventful surgery, and no significant differences between groups were observed during the clin. course. Multiple statistical analyses with different methods were performed. Compared with preoperative expression at a threshold value of $P < 0.01$, postoperative expression revealed 814 upregulated and 1187 downregulated genes in group H compared with 99 upregulated and 231 downregulated in group P. Fifty genes exhibited a >4-fold increase and 27 exhibited a >4-fold decrease in group H, whereas only 7 genes exhibited upregulation and 7 revealed downregulation in group P. Microarray-pathway-profile-finder analyses determined 1405 upregulated and 1454 downregulated pathways in group H compared with 552 upregulated and 818 downregulated pathways in group P. Pathways related to inflammatory response exhibited highest z scores in group H, reflecting cellular inflammatory activation. Conclusions-Heparin coating resulted in a more profound alteration in leukocyte gene expression when compared with protein coating. Microarray analyses present an innovative approach for the evaluation and understanding of inflammatory reactions associated with CPB.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 45 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:878417 CAPLUS Full-text

DOCUMENT NUMBER: 141:362216

TITLE: Blood plasma peptides, including human secretory leukocyte protease inhibitor, associated with coronary artery disease and their diagnostic and therapeutic uses

INVENTOR(S): Argoud-Puy, Guilaine; Bederr, Nassima; Bougueleret, Lydie; Cusin, Isabelle; Mahe, Eve; Niknejad, Anne; Reffas, Samia

PATENT ASSIGNEE(S): Genova Ltd., Bermuda; Novartis Pharma GmbH

SOURCE: PCT Int. Appl., 145 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2004089987	A1	20041021	WO 2004-EP3746	20040407
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1613653	A1	20060111	EP 2004-726158	20040407
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				

JP 2007534611 T 20071129 JP 2006-505056 20040407
 US 20070098635 A1 20070503 US 2006-552401 20061205
 PRIORITY APPLN. INFO.: US 2003-461560P P 20030408
 WO 2004-EP3746 W 20040407

ED Entered STN: 22 Oct 2004

AB The invention discloses human secreted polypeptides that circulate at an increased level in the plasma of patients with cardiovascular disorders. Polypeptides of the invention comprise CPP (cardiovascular disorder plasma polypeptide). Two peptides are tryptic fragments of antileukoproteinase (ALP), also called secretory leukocyte protease inhibitor. CPP8 (SEQ ID:2) is a mature form of antileukoproteinase. The invention also provides methods of using compns. including the polypeptides, polynucleotides encoding them, and antibodies specific for these polypeptides, for diagnosis, prognosis, and treatment of cardiovascular disorders.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 46 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:878267 CAPLUS Full-text

DOCUMENT NUMBER: 141:360719

TITLE: Immunomodulatory proteins for treatment of inflammatory diseases

INVENTOR(S): Palefsky, Joel; Sroussi, Herve

PATENT ASSIGNEE(S): The Regents of the University of California, USA

SOURCE: PCT Int. Appl., 51 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004089292	A2	20041021	WO 2004-US10244	20040402
WO 2004089292	A3	20060629		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 20070123455 A1 20070531 US 2007-551704 20070122
 PRIORITY APPLN. INFO.: US 2003-460652P P 20030404
 WO 2004-US10244 W 20040402

ED Entered STN: 22 Oct 2004

AB The present invention provides methods and compns. suitable for treating inflammatory disorders such as allergy, asthma, arteriosclerosis, autoimmune disease, infection, injury, meningitis, psoriasis, and transplant rejection. In particular, the present invention provides methods and compns. comprising human S100A8 and/or S100A9 protein for reducing inflammation. For example, S100A9 by itself or complexed to S100A8 was found to enhance the growth of Pseudomonas aeruginosa in vitro. The 61ALA mutant of S100A9 with substitutions of methionine 61, 81 and 83 with alanine, did not induce this enhanced growth, and even competed with the wild type protein. Thus, modified S100A9 proteins are contemplated to be suitable therapeutics for Pseudomonas-associated pneumonia, especially for treatment of Pseudomonas-induced pneumonia in patients with cystic fibrosis, as these patients have high levels

of calprotectin (wild type S100A8/A9 complexes) in their pulmonary secretions. The mutants could be used alone or in conjunction with standard anti-pseudomonal antibiotics. In addition, the ALA42-S100A8 mutant is also contemplated to act as an inhibitor of the wild-type S100A9 and S100A8/A9-induced growth of Pseudomonas. In short, S100A8/A9 enhancement of Pseudomonas growth is contemplated to be partly responsible for the recurrent Pseudomonas infections observed in cystic fibrosis patients. The ALA42-S100A8 and/or the 61ALA-S100A9 proteins are contemplated to be suitable therapeutic proteins against Pseudomonas lung infection on their own, or in conjunction with other available therapeutics.

L7 ANSWER 47 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN
 ACCESSION NUMBER: 2004:546624 CAPLUS Full-text
 DOCUMENT NUMBER: 141:67871
 TITLE: Cardiovascular disease assay
 INVENTOR(S): Sundrehagen, Erling
 PATENT ASSIGNEE(S): Axis-Shield Asa, Norway; Cockbain, Julian
 SOURCE: PCT Int. Appl., 58 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004057341	A2	20040708	WO 2003-GB5607	20031222
WO 2004057341	A3	20041021		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2504876	A1	20040708	CA 2003-2504876	20031222
AU 2003295142	A1	20040714	AU 2003-295142	20031222
EP 1573335	A2	20050914	EP 2003-786143	20031222
EP 1573335	B1	20071205		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1729398	A	20060201	CN 2003-80106912	20031222
JP 2006510895	T	20060330	JP 2004-561672	20031222
EP 1739430	A2	20070103	EP 2006-21890	20031222
EP 1739430	A3	20070117		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LI, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AT 380345	T	20071215	AT 2003-786143	20031222
ES 2297250	T3	20080501	ES 2003-786143	20031222
NO 2005002161	A	20050719	NO 2005-2161	20050503
US 20060134705	A1	20060622	US 2005-539797	20051219
PRIORITY APPLN. INFO.:			GB 2002-29747	A 20021220
			EP 2003-786143	A3 20031222
			WO 2003-GB5607	W 20031222

ED Entered STN: 08 Jul 2004

AB An assay method for the detection of potential for CVD or propensity to CVD in a human or non-human animal subject, said method comprising assessing the

concentration of calprotectin in a calprotectin containing sample taken from said subject.

L7 ANSWER 48 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:525453 BIOSIS Full-text
DOCUMENT NUMBER: PREV200510315416
TITLE: Possible role of myeloid related protein (MRP) 8 and MRP14 in
progression of coronary artery lesions in acute Kawasaki disease.
AUTHOR(S): Hirono, Keiichi [Reprint Author]; Ye, Fei; Foell, Dirk;
Watanabe, Kazuhiro; Watanabe, Sayaka; Uese, Keiichirou; Roth, Johannes; Miyawaki,
Toshio; Ichica, Fukiko
CORPORATE SOURCE: Univ Munster, D-4400 Munster, Germany
SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 387.
Meeting Info.: 77th Scientific Meeting of the American-Heart-
Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005
ED Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005
AB Myeloid related Protein (MRP) 8 and MRP14, S100-proteins secreted by activated
neutrophils and monocytes, bind specifically to endothelial cells, and induce
thrombogenic and inflammatory response in a variety of disease conditions.
Kawasaki disease (KD) is the most common systemic vasculitis syndrome
primarily affecting small and medium-sized arteries, particularly the coronary
artery. Histopathological studies Have shown that the coronary microvascular
lesions were characterized by endothelial cell injury and platelet aggregation
with thrombosis. The aim of our study was to investigate the role of
MRP8/MRP14 in acute KD, and to analyse the usefulness of its serum levels for
monitoring severity of vasculitis. Methods: We investigated 78 patients with
acute KD differentiated by complication of coronary artery lesion (CAL). The
serum levels of MRP8/MRP14 were determined by ELISA. Results: The vast
majority of KID patients in the acute phase showed higher serum MRP8/MRP14
levels than normal controls. In the group of 65 patients without CAL, serum
MRP8/MRP14 decreased dramatically within 24 hours of IVIG therapy (mean
difference 1250 ng/ml, p=0.01). In contrast, 13 patients with CAL exhibited
the increment of serum MRP8/MRP14 levels 24 h after IVIG therapy, but
decreased after retreatment and achieved its attenuation by 4 weeks post-
onset. The patients with CAL had higher maximum concentrations of MRP8/MRP14
than those without (mean difference 1580 ng/ml, P=0.002). Minimum platelet
counts were lower in non-responders to IVIG therapy, as compared to responders
(p<0.01). Interestingly, minimum platelets counts were closely correlated
with maximum serum levels of MRP8/14 (p<0.05). Conclusions: The present
study suggested that MRP8/MRP14 expression closely correlated with disease
activity of acute KID, and might provide a useful serum marker in monitoring
severity of vasculitis. In addition, MRP8/14 might induce a thrombogenic
response in microvascular wall in acute KD, Our data imply an important
functional role of MRP8/14 and might open a perspective for the development of
blocking agents for the use in human vasculitis syndromes.

L7 ANSWER 49 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 17
ACCESSION NUMBER: 2003:942767 CAPLUS Full-text
DOCUMENT NUMBER: 140:40262
TITLE: Genes expressed in atherosclerotic tissue and their use in
diagnosis and pharmacogenetics

INVENTOR(S): Nevins, Joseph; West, Mike; Goldschmidt, Pascal
 PATENT ASSIGNEE(S): Duke University, USA
 SOURCE: PCT Int. Appl., 408 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 5
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003091391	A2	20031106	WO 2002-XB38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003091391	A2	20031106	WO 2002-US38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:
 US 2002-374547P P 20020423
 US 2002-420784P P 20021024
 US 2002-421043P P 20021025
 US 2002-424680P P 20021108
 WO 2002-US38221 A 20021112

ED Entered STN: 04 Dec 2003

AB Genes whose expression is correlated with an determinant of an atherosclerotic phenotype are provided. Also provided are methods of using the subject atherosclerotic determinant genes in diagnosis and treatment methods, as well as drug screening methods. In addition, reagents and kits thereof that find use in practicing the subject methods are provided. Also provided are methods of determining whether a gene is correlated with a disease phenotype, where correlation is determined using a Bayesian anal. [This abstract record is one of three records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.]

L7 ANSWER 50 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:657020 CAPLUS Full-text

DOCUMENT NUMBER: 139:193985

TITLE: Method of diagnosis of inflammatory diseases using calgranulin C and treatment of the diseases based on stage of disease

INVENTOR(S): Sorg, Clemens; Roth, Johannes

PATENT ASSIGNEE(S): Switch Biotech A.-G., Germany

SOURCE: PCT Int. Appl., 64 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	-----	-----	-----	-----
WO 2003069341	A2	20030821	WO 2003-EP1575	20030217
WO 2003069341	A3	20040205		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 20030175713	A1	20030918	US 2002-77600	20020215
CA 2474890	A1	20030821	CA 2003-2474890	20030217
AU 2003212245	A1	20030904	AU 2003-212245	20030217
EP 1474689	A2	20041110	EP 2003-708103	20030217
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005517414	T	20050616	JP 2003-568411	20030217
US 20050147972	A1	20050707	US 2005-504299	20050218
PRIORITY APPLN. INFO.:			US 2002-77600	A 20020215
			WO 2003-EP1575	W 20030217

ED Entered STN: 22 Aug 2003

AB The present invention is directed to a method for diagnosing inflammatory diseases based on the marker calgranulin C, particularly for diagnosing specific stages of inflammatory diseases and/or for determining the risk of relapse and/or for discriminating between diseases with similar symptoms, said method comprising the steps of (a) obtaining a biol. sample of mammalian body fluid or tissue to be diagnosed; (b) determining the amount and/or concentration of calgranulin C polypeptide and/or nucleic acids encoding the polypeptide present in said biol. sample; and (c) comparing the amount and/or concentration of calgranulin C polypeptide determined in said biol. sample with the amount and/or concentration of calgranulin C polypeptide as determined in a control sample and/or comparing the amount and/or concentration of nucleic acids encoding calgranulin C polypeptide determined in said biol. sample with the amount and/or concentration of nucleic acids encoding calgranulin C polypeptides measured in a control sample, wherein the difference in the amount of calgranulin C polypeptide and/or nucleic acids encoding the polypeptide is indicative for the stages of the disease to be diagnosed. Concns. of calgranulin C in the serum of patients were determined by a double sandwich ELISA. Cystic fibrosis patients with acute exacerbation had significantly elevated levels.

L7 ANSWER 51 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:598234 CAPLUS Full-text

DOCUMENT NUMBER: 139:305614

TITLE: Brief episode of ischemia activates protective genetic program in rat heart: a gene chip study

AUTHOR(S): Simkhovich, Boris Z.; Marjoram, Paul; Poizat, Coralie; Kedes, Larry; Kloner, Robert A.

CORPORATE SOURCE: Heart Institute, Good Samaritan Hospital, Los Angeles, CA, 90017, USA

SOURCE: Cardiovascular Research (2003), 59(2), 450-459
CODEN: CVREAU; ISSN: 0008-6363

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 05 Aug 2003

AB Objective: Brief episodes of ischemia of 20 min or less have the potential to protect the heart. Such episodes are associated primarily with reversible ischemic injury yet they induce changes in gene expression. The purpose of the study was to determine whether activation of protective genes takes place within 4 h following a brief episode of ischemia that would mimic angina pectoris. Methods: Three groups of rats were studied. In the control (Ctrl) group, hearts were immediately excised following anesthesia; in the sham-operated (SO) group, opened-chest rats received 4 h and 20 min of no intervention; and in the group subjected to ischemia (SI) hearts received 20 min of proximal coronary occlusion followed by 4 h of reperfusion. Hearts from the SI group were divided into nonischemic (NI) and ischemic (Isc) areas. Changes in gene expression pattern were analyzed by using Affymetrix Gene Chips. Results: Ischemia led to strong upregulation of mRNA transcripts for heat shock proteins 70, 27, 105, 86 and 40 kDa, vascular endothelial growth factor, brain-derived neurotrophic factor, plasminogen activator inhibitor-1, activating transcription factor 3, B-cell translocation gene 2, and growth arrest and DNA damage inducible 45 α protein compared to the NI tissue. The majority of mRNAs whose levels increased following brief ischemia were of a protective nature. Conclusion: Genetic reprogramming emerging during or following brief episodes of ischemia that simulate angina, can be characterized as protective in nature. Developing new therapeutic strategies aimed to promote this protective response represents a legitimate target for future research.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 52 OF 68 MEDLINE on STN
ACCESSION NUMBER: 2002372602 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12118194
TITLE: Phenotypic characterization of infiltrates in dilated cardiomyopathy - diagnostic significance of T-lymphocytes and macrophages in inflammatory cardiomyopathy.
AUTHOR: Noutsias Michel; Pauschinger Matthias; Schultheiss Heinz; K h1 Uwe
CORPORATE SOURCE: Department of Cardiology and Pneumology, University Hospital Benjamin Franklin, Free University of Berlin, Germany.. noutsias@zedat.fu-berlin.de
SOURCE: Medical science monitor : international medical journal of experimental and clinical research, (2002 Jul) Vol. 8, No. 7, pp. CR478-87.
Journal code: 9609063. ISSN: 1234-1010.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 16 Jul 2002
Last Updated on STN: 19 Mar 2003
Entered Medline: 18 Mar 2003

ED Entered STN: 16 Jul 2002
Last Updated on STN: 19 Mar 2003
Entered Medline: 18 Mar 2003

AB BACKGROUND: Dilated cardiomyopathy (DCM) is linked to inflammatory cardiomyopathy (InfCM). To gain a profound insight into the underlying mechanisms, we phenotypically characterized pan leukocytes (CD18), naive T-lymphocytes (CD3, CD2, CD4, CD8), activated lymphocytes (LFA-1, LFA-3, VLA-4, ICAM-1, CD69, CD45RO), macrophages (Mac-1, 27E10), B-lymphocytes (CD19) and NK-cells (CD57) in DCM and control hearts. MATERIAL/METHODS: Biopsies from DCM patients (n=164, LVEF<40%) and specimens from non-cardiac death causes (controls; n=17) were immunostained. Biopsies exceeding >2.0 CD3+ lymphocytes per high power field/hpf and/or >1.5 CD3+ lymphocytes/hpf with numerous foci

and HLA class I/DR abundance were evaluated positive for InfCM. RESULTS: InfCM+ biopsies (n=102; 63%) demonstrated significantly increased infiltrates with respect to all studied phenotypes except for CD19 and CD57 when compared with both DCM biopsies negative for InfCM (n=62) and the controls, whereas the latter two groups did not differ (Tukey-Kramer analysis). Virtually all phenotypes correlated with one another in multivariate analysis (except for B-lymphocytes and NK cells). Whereas HLA class I/DR abundance was present in 14% of the controls and 26% of the DCM biopsies not yielding InfCM, InfCM+ biopsies demonstrated significantly (<0.001) higher frequencies of HLA abundance (76%). CONCLUSIONS: The inflammatory process in InfCM comprises T-lymphocytes and macrophages, whereas B-lymphocytes and NK-cells are not significantly increased. InfCM is associated with HLA induction. CD69+, CD45R0+ and adhesion molecule bearing infiltrates indicate the activated state of lymphocytes, and 27E10 of macrophages in InfCM, respectively. Our data are in accordance with the hypothesis of a 'chronic active inflammatory process' involved in DCM.

L7 ANSWER 53 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2002:665693 CAPLUS Full-text

DOCUMENT NUMBER: 138:151329

TITLE: Profile of crucial gene expression to cardioprotective effects of ischemic preconditioning: using high-density oligonucleotide array technology

AUTHOR(S): Miura, Nobuko

CORPORATE SOURCE: Department of Cardiology, Kanazawa Medical University, Ishikawa, 920-0293, Japan

SOURCE: Kanazawa Ika Daigaku Zasshi (2002), 27(1), 9-17
CODEN: KIDZDN; ISSN: 0385-5759

PUBLISHER: Kanazawa Ika Daigaku Shuppan Kyoku

DOCUMENT TYPE: Journal

LANGUAGE: Japanese

ED Entered STN: 04 Sep 2002

AB Ischemic Preconditioning (IPC) of the heart with brief periods of ischemia/reperfusion injury protects against subsequent sustained ischemia-reperfusion. In the present study, gene expression profile of myocardium with or without IPC was investigated using high-d. oligonucleotide array (GeneChip) technol. Male Wistar rats were assigned into the following three groups: sham operation (sham group), ischemia/reperfusion (IR group), and IPC procedure before ischemia/reperfusion (IPC-IR group). Except the sham group, all rats underwent sustained coronary artery occlusion (CAO) for 30 min followed by 180 min of reperfusion. IPC was elicited with 4 cycles of 5 min ischemia and 5 min reperfusion before CAO. Thirteen genes (Mn-SOD, junB, B cell translocation, MRP8 calcium-binding protein, MRP14 calcium-binding protein, insulin-like growth factor-binding protein, Egr-1, IgE binding protein, pyruvate dehydrogenase kinase, c-fos, gene33, HO-1 genes, and DNA damage-inducible transcript) were significantly up-regulated by IR, and further up-regulated by IPC-IR. In contrast, two genes (HO-3 and heat shock protein) were revealed to be up-regulated only by IR. Five genes (fatty acid transporter, Jak 1, tyrosine phosphatase, sodium/potassium transporting ATPase and VEGF) were down-regulated by I/R. Up-regulation of 8 genes (Mn-SOD, MRP8, MRP14, g33, c-fos, Egr-1, HO-1 and VEGF) was confirmed by reverse-transcriptase PCR (RT-PCR). To eliminate the effect of non-myocardial cells, changes in above gene expression, cardiac myocytes cultured in new born rats were investigated by RT-PCR under conditioned normoxia, hypoxia (3 h hypoxia) and IPC (30 min normoxia and 30 min hypoxia before 3 h hypoxia), resp. Gene expressions of Erg-1, HO-1, VEGF and g33 showed a similar response to hypoxia or IPC as observed in vivo study. These results indicated that a cluster of the same genes might play significant roles in myocardial IPC.

L7 ANSWER 54 OF 68 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002272682 EMBASE Full-text

TITLE: Phenotypic characterization of infiltrates in dilated cardiomyopathy - Diagnostic significance of T-lymphocytes and macrophages in inflammatory cardiomyopathy.

AUTHOR: Noutsias, Michel, Dr. (correspondence); Pauschinger, Matthias; Schultheiss, Heinz-Peter; Kuhl, Uwe

CORPORATE SOURCE: Department of Cardiology, Univ. Hospital Benjamin Franklin, Free University of Berlin, Hindenburgdamm 30, D-12200 Berlin, Germany.

noutsias@zedat.fu-berlin.de

SOURCE: Medical Science Monitor, (2002) Vol. 8, No. 7, pp. CR478-CR487.

Refs: 55

ISSN: 1234-1010 CODEN: MSMOFR

COUNTRY: Poland

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
005 General Pathology and Pathological Anatomy

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 15 Aug 2002

Last Updated on STN: 15 Aug 2002

ED Entered STN: 15 Aug 2002

Last Updated on STN: 15 Aug 2002

AB Background: Dilated cardiomyopathy (DCM) is linked to inflammatory cardiomyopathy (InfCM). To gain a profound insight into the underlying mechanisms, we phenotypically characterized pan leukocytes (CD18), naive T-lymphocytes (CD3, CD2, CD4, CD8), activated lymphocytes (LFA-1, LFA-3, VLA-4, ICAM-1, CD69, CD45RO), macrophages (Mac-1, 27E10), B-lymphocytes (CD19) and NK-cells (CD57) in DCM and control hearts. Material/Methods: Biopsies from DCM patients (n=164, LVEF<40%) and specimens from non-cardiac death causes (controls; n=17) were immunostained. Biopsies exceeding >2.0 CD3+ lymphocytes per high power field/hpf and/or >1.5 CD3+ lymphocytes/hpf with numerous foci and HLA class I/DR abundance were evaluated positive for InfCM. Results: InfCM+ biopsies (n= 102; 63%) demonstrated significantly increased infiltrates with respect to all studied phenotypes except for CD19 and CD57 when compared with both DCM biopsies negative for InfCM (n=62) and the controls, whereas the latter two groups did not differ (Tukey-Kramer analysis). Virtually all phenotypes correlated with one another in multivariate analysis (except for B-lymphocytes and NK cells). Whereas HLA class I/DR abundance was present in 14% of the controls and 26% of the DCM biopsies not yielding InfCM, InfCM+ biopsies demonstrated significantly (<0.001) higher frequencies of HLA abundance (76%). Conclusions: The inflammatory process in InfCM comprises T-lymphocytes and macrophages, whereas B-lymphocytes and NK-cells are not significantly increased. InfCM is associated with HLA induction. CD69+, CD45RO+ and adhesion molecule bearing infiltrates indicate the activated state of lymphocytes, and 27E10 of macrophages in InfCM, respectively. Our data are in accordance with the hypothesis of a 'chronic active inflammatory process' involved in DCM.

L7 ANSWER 55 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:678594 CAPLUS Full-text

DOCUMENT NUMBER: 135:328533

TITLE: Novel intra- and inter-molecular sulfinamide bonds in S100A8 produced by hypochlorite oxidation

AUTHOR(S): Raftery, Mark J.; Yang, Zheng; Valenzuela, Stella M.; Geczy, Carolyn L.

CORPORATE SOURCE: Cytokine Research Unit, School of Pathology, University of
New South Wales, Kensington, 2052, Australia
SOURCE: Journal of Biological Chemistry (2001), 276(36), 33393-
33401

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 17 Sep 2001

AB Hypochlorite is a major oxidant generated when neutrophils and macrophages are activated at inflammatory sites, such as in atherosclerotic lesions. Murine S100A8 (A8) is a major cytoplasmic protein in neutrophils and is secreted by macrophages in response to inflammatory stimuli. After incubation with reagent HOCl for 10 min, .apprx.85% of A8 was converted to 4 oxidation products, with electrospray ionization mass spectrometry masses of m/z 10354, 10388, 10354±1, and 20707±3. All were resistant to reduction by dithiothreitol. Initial formation of a reactive Cys sulfenic acid intermediate was demonstrated by the rapid conjugation of 5,5-dimethyl-1,3-cyclohexanedione (dimedone) to HOCl-treated A8 to form stable adducts. Matrix-assisted laser desorption-reflection time of flight peptide mass fingerprinting of isolated oxidation products confirmed the mass addns. observed in the full-length proteins. Both Met36/73 were converted to Met36/73 sulfoxides. An addnl. product with an unusual mass addition of m/z 14 (±0.2) was identified and corresponded to the addition of oxygen to Cys41, conjugation to various ε-amines of Lys6, Lys34/35, or Lys87 with loss of dihydrogen and formation of stable intra- or inter-mol. sulfinamide cross-links. Specific fragmentations identified in matrix-assisted laser desorption-post source decay spectra and low energy collisional-induced dissociation tandem mass spectroscopy spectra of sulfinamide-containing digest peptides confirmed Lys34/35 to Cys41 sulfinamide bonds. HOCl oxidation of mutants lacking Cys41 (Ala41S100A8) or specific Lys residues (e.g. Lys34/35, Ala34/35S100A8) did not form sulfinamide cross-links. HOCl generated by myeloperoxidase and H2O2 and by phorbol 12-myristate 13-acetate-activated neutrophils also formed these products. In contrast to the disulfide-linked dimer, oxidized monomer retained normal chemotactic activity for neutrophils. Sulfinamide bond formation represents a novel oxidative crosslinking process between thiols and amines and may be a general consequence of HOCl protein oxidation in inflammation not identified previously. Similar modifications in other proteins could potentially regulate normal and pathol. processes during aging, atherogenesis, fibrosis, and neurogenerative diseases.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 56 OF 68 MEDLINE on STN DUPLICATE 18

ACCESSION NUMBER: 2001382772 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 11295086

TITLE: Immunohistochemical investigation of S100A9 expression in
pulmonary adenocarcinoma: S100A9 expression is associated with tumor
differentiation.

AUTHOR: Arai K; Teratani T; Nozawa R; Yamada T

CORPORATE SOURCE: Department of Pathology, Shizuoka General Hospital, Shizuoka
420-0881, Japan.. m-arai@ny.tokai.or.jp

SOURCE: Oncology reports, (2001 May-Jun) Vol. 8, No. 3, pp. 591-6.
Journal code: 9422756. ISSN: 1021-335X.

PUB. COUNTRY: Greece

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200107

ENTRY DATE: Entered STN: 9 Jul 2001

Last Updated on STN: 9 Jul 2001

Entered Medline: 5 Jul 2001

ED Entered STN: 9 Jul 2001

Last Updated on STN: 9 Jul 2001

Entered Medline: 5 Jul 2001

AB S100 protein A9 is associated with myeloid cell differentiation and is also expressed in some epithelia. However, there have been few studies on S100A9 in specific types of carcinomas, except for squamous cell carcinoma (SCC) because the expression in normal epithelia is limited to squamous epithelia. Recently, S100A9 gene expression has been detected in cultured human adenocarcinoma (AC) cells derived from various organs. In this study, we also detected S100A9 gene expression in human pulmonary AC cell lines by reverse transcription-polymerase chain reaction. Furthermore, using the monoclonal antibody against S100A9, we carried out an immunohistochemical evaluation of S100A9 protein expression in 70 cases of resected pulmonary AC and examined the relation of S100A9 expression to tumor differentiation. S100A9 immunopositivity was 0/21 (0%) in well differentiated ACs, 12/30 (40%) in moderately differentiated ACs and 19/19 (100%) in poorly differentiated ACs, and the poorly differentiated ACs showed a significantly greater positive reaction. The immunopositivity in the moderately differentiated ACs was marked in specific cytologic subtypes. In the controls, conspicuous S100A9 immunopositivity was observed in pulmonary SCCs, regardless of the degree of differentiation, but not in adenomatous hyperplasia or normal surface epithelia. These above results suggest that the S100A9 protein is also expressed in pulmonary AC and that the expression rate in pulmonary AC shows higher correlation in poorly differentiated carcinomas, in agreement with our recent results regarding liver carcinoma. We believe S100A9 is also closely related to the differentiation of carcinomas of glandular cell origin.

L7 ANSWER 57 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:142577 CAPLUS Full-text

DOCUMENT NUMBER: 134:309006

TITLE: Arachidonic acid specifically regulates binding of S100A8/A9, a heterodimer complex of the S100 class of calcium binding proteins, to human microvascular endothelial cells

AUTHOR(S): Eue, I.; Sorg, C.

CORPORATE SOURCE: Institute of Experimental Dermatology, University of Munster, Munster, 48149, Germany

SOURCE: Atherosclerosis (Shannon, Ireland) (2001), 154(2), 505-508
CODEN: ATHSBL; ISSN: 0021-9150

PUBLISHER: Elsevier Science Ireland Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 28 Feb 2001

AB The authors data again support the physiolo. relevance and importance of the S100A8/A9-arachidonic acid complex formation not only in the context of neutrophil activation, but also in the setting of activated endothelium as it occurs both in inflammatory and proatherogenic processes.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 58 OF 68 MEDLINE on STN DUPLICATE 19

ACCESSION NUMBER: 2002078171 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 11803621

TITLE: MRP 8/14 and procalcitonin serum levels in organ transplantations.

AUTHOR: Striz I; Jaresova M; Lacha J; Sedlacek J; Vitko S

CORPORATE SOURCE: Institute for Clinical and Experimental Medicine, Prague, Czech Republic.

SOURCE: Annals of transplantation : quarterly of the Polish
Transplantation Society, (2001) Vol. 6, No. 2, pp. 6-9.
Journal code: 9802544. ISSN: 1425-9524.

PUB. COUNTRY: Poland

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200207

ENTRY DATE: Entered STN: 28 Jan 2002

Last Updated on STN: 27 Jul 2002

Entered Medline: 11 Jul 2002

ED Entered STN: 28 Jan 2002

Last Updated on STN: 27 Jul 2002

Entered Medline: 11 Jul 2002

AB OBJECTIVES: MRP8/14 is a heterodimer of two myeloid calcium-binding proteins associated with different types of acute inflammatory processes. We studied MRP8/14 together with procalcitonin (PCT) serum levels in order to diagnose infectious complications or the rejection process affecting kidney or heart allograft. METHODS: A total of 419 serum samples was evaluated. MRP8/14 levels were measured by ELISA (BMA Biomed), PCT by a sensitive immunoluminiscent assay ILMA (Brahms Diagn.) RESULTS: Both parameters showed very low basal levels in healthy subjects (range 303-1,660 ng/ml of MRP8/14; less than 0.08 ng/ml of PCT). A rapid increase in serum levels occurred in response to bacterial infections (MRP8/14 up to 6,230 ng/ml; PCT up to 297 ng/ml). Serum PCT concentration remained low in the presence of kidney allograft rejection, where MRP8/14 levels were increased. An uncomplicated outcome of kidney or heart transplantation did not change basal serum MRP8/14 and PCT levels. CONCLUSIONS: We conclude that 1) both MRP8/14 and PCT are very sensitive markers of complications in organ transplant recipients (normal values in uncomplicated outcome) 2) combination of both parameters is useful to discriminate between rejection (increased MRP8/14 with normal PCT) and systemic bacterial infection (both parameters increased).

L7 ANSWER 59 OF 68 MEDLINE on STN DUPLICATE 20

ACCESSION NUMBER: 2001029643 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 10944082

TITLE: Myeloid related protein (MRP) 14 expressing monocytes
infiltrate atherosclerotic lesions of ApoE null mice.

AUTHOR: Eue I; Langer C; Eckardstein A; Sorg C

SOURCE: Atherosclerosis, (2000 Aug) Vol. 151, No. 2, pp. 593-7.
Journal code: 0242543. ISSN: 0021-9150.

PUB. COUNTRY: Ireland

DOCUMENT TYPE: Letter

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200011

ENTRY DATE: Entered STN: 22 Mar 2001

Last Updated on STN: 22 Mar 2001

Entered Medline: 21 Nov 2000

ED Entered STN: 22 Mar 2001

Last Updated on STN: 22 Mar 2001

Entered Medline: 21 Nov 2000

L7 ANSWER 60 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2000:742237 CAPLUS Full-text

DOCUMENT NUMBER: 133:291117

TITLE: Use of peptides of S100 proteins in the treatment of heart
failure

INVENTOR(S): Katus, Hugo A.; Remppis, Andrew
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000061742	A2	20001019	WO 2000-EP2453	20000320
WO 2000061742	A3	20010329		
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
DE 19915485	A1	20001019	DE 1999-19915485	19990407
CA 2369826	A1	20001019	CA 2000-2369826	20000320
EP 1169441	A2	20020109	EP 2000-916980	20000320
EP 1169441	B1	20050608		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002542168	T	20021210	JP 2000-611666	20000320
AT 297464	T	20050615	AT 2000-916980	20000320
ES 2243246	T3	20051201	ES 2000-916980	20000320
PRIORITY APPLN. INFO.:			DE 1999-19915485	A 19990407
			WO 2000-EP2453	W 20000320

ED Entered STN: 20 Oct 2000

AB The invention relates to medicaments for treating cardiac power failure. Said medicaments contain a therapeutically effective quantity of one or more S100 protein(s) or one or more mutant or fragments of the same, or contain one or more nucleic acid sequence(s) which code(s) for these amino acid sequences and which are optionally integrated in one or more gene transfer vectors. The condition may be treated by administration of the peptides or by gene therapy using expression constructs for manufacture of the peptides in situ. Human S-100A1 was manufactured by expression of the protein in Escherichia coli. Incubation of fetal rat cardiomyocytes with the protein increased the amplitude of Ca²⁺ transients in the sarcoplasmic reticulum. Rabbits inoculated with an adenovirus vector expressing the S-100A1 gene showed an increased systolic ejection pressure. Control animals inoculated with vector only showed a drop in systolic pressure.

L7 ANSWER 61 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2000:560481 CAPLUS Full-text

DOCUMENT NUMBER: 133:221057

TITLE: Myeloid related protein (MRP) 14 expressing monocytes infiltrate atherosclerotic lesions of ApoE null mice

AUTHOR(S): Eue, I.; Langer, C.; v. Eckardstein, A.; Sorg, C.

CORPORATE SOURCE: Institute of Experimental Dermatology, University of Munster, Munster, Germany

SOURCE: Atherosclerosis (Shannon, Ireland) (2000), 151(2), 593-597
CODEN: ATHSBL; ISSN: 0021-9150

PUBLISHER: Elsevier Science Ireland Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 15 Aug 2000

AB The authors investigated the cellular infiltration of atherosclerotic lesions at different time points and the functional characterization of these cells. Myeloid related protein 14 (MRP14) expressing cells were found in the atherosclerotic lesions of 6 mo old ApoE -/- mice, where MRP8 pos. cells very

rarely occurred in the plaques. MRP14 expressing cells were identified as monocytes.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 62 OF 68 MEDLINE on STN

ACCESSION NUMBER: 2001393212 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 11122775

TITLE: Atherosclerosis and diabetes: the RAGE connection.

AUTHOR: Schmidt A M; Stern D

CORPORATE SOURCE: Department of Surgery, College of Physicians & Surgeons of Columbia University, 630 West 168th Street, New York, New York 10032, USA.

SOURCE: Current atherosclerosis reports, (2000 Sep) Vol. 2, No. 5, pp. 430-6. Ref: 37

Journal code: 100897685. ISSN: 1523-3804.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200107

ENTRY DATE: Entered STN: 16 Jul 2001

Last Updated on STN: 16 Jul 2001

Entered Medline: 12 Jul 2001

ED Entered STN: 16 Jul 2001

Last Updated on STN: 16 Jul 2001

Entered Medline: 12 Jul 2001

AB Diabetes mellitus begins as a disorder of glucose metabolism that progressively compromises the function of virtually every organ system as the secondary complications inexorably develop. The quality of life for patients with diabetes is diminished by the consequences of these complications. Accelerated and aggressive atherosclerosis is the greatest cause of morbidity and mortality with diabetes, emphasizing the importance of determining underlying mechanisms. This review highlights the role of the multiligand receptor for advanced glycation endproducts (RAGE) and two of its ligands, advanced glycation endproducts (AGEs) and S100/calgranulins, in the pathogenesis of atherosclerosis associated with diabetes. The results of the studies reviewed herein suggest that RAGE is a potential therapeutic target for macrovascular disease in diabetes.

L7 ANSWER 63 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:5060 BIOSIS Full-text

DOCUMENT NUMBER: PREV199799304263

TITLE: Calprotectin: A novel plasma marker of granulocyte activation in acute coronary syndrome.

AUTHOR(S): Arvesen, Kristin; Kontny, Frederic; Toss, Henrik; Wallentin, Lars; Roseth, Arne; Sundset, Alison; Fagerhol, Magne K.

CORPORATE SOURCE: Aker Univ. Hosp., Oslo, Norway

SOURCE: Circulation, (1996) Vol. 94, No. 8 SUPPL., pp. I514.

Meeting Info.: 69th Scientific Sessions of the American Heart Association. New Orleans, Louisiana, USA. November 10-13, 1996.

CODEN: CIRCAZ. ISSN: 0009-7322.

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 7 Jan 1997

Last Updated on STN: 7 Jan 1997

ED Entered STN: 7 Jan 1997
Last Updated on STN: 7 Jan 1997

L7 ANSWER 64 OF 68 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:561116 BIOSIS Full-text
DOCUMENT NUMBER: PREV199699283472
TITLE: Calprotectin: A novel plasma marker of granulocyte activation
in acute coronary syndrome.
AUTHOR(S): Arvesen, K. [Reprint author]; Kontny, F. [Reprint author];
Toss, H.; Wallentin, L.; Roseth, A. [Reprint author]; Sundset, A.; Fagerhol, M. K.
CORPORATE SOURCE: Dep. Med., Aker Univ. Hosp., Oslo, Norway
SOURCE: European Heart Journal, (1996) Vol. 17, No. ABSTR. SUPPL., pp.
429.

Meeting Info.: XVIIIth Congress of the European Society of
Cardiology. Birmingham, England, UK. August 25-29, 1996.

CODEN: EHJODF. ISSN: 0195-668X.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 13 Dec 1996

Last Updated on STN: 13 Dec 1996

ED Entered STN: 13 Dec 1996
Last Updated on STN: 13 Dec 1996

L7 ANSWER 65 OF 68 MEDLINE on STN DUPLICATE 21
ACCESSION NUMBER: 1997010637 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 8857675
TITLE: Release of interleukin-8 and calprotectin during and after
paediatric cardiopulmonary bypass with and without ultrafiltration.
AUTHOR: Saatvedt K; Lindberg H; Geiran O R; Michelsen S; Pedersen T;
Seem E; Fagerhol M
CORPORATE SOURCE: Department of Cardiovascular Surgery, Rikshospitalet, Oslo,
Norway.
SOURCE: Scandinavian journal of thoracic and cardiovascular surgery,
(1996) Vol. 30, No. 2, pp. 53-9.
Journal code: 0121343. ISSN: 0036-5580.
PUB. COUNTRY: Sweden
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 7 Jan 1997

ED Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 7 Jan 1997

AB Release of calprotectin and interleukin-8 (IL-8), changes in leukocyte counts
and subsets and influence of extracorporeal ultrafiltration were evaluated
during and after cardiopulmonary bypass (CPB) in 18 children undergoing open-
heart surgery for congenital heart anomalies. Ultrafiltration was used in
nine cases and nine were controls. Calprotectin concentration rose after
start of CPB, peaking 48 hours postoperatively, with no significant intergroup
difference. Positive correlation was found between duration of CPB and
calprotectin (peak level and accumulated total). Circulating IL-8 was
detected in all patients perioperatively, peaking at wound closure in the
ultrafiltration group and at termination of bypass in the controls. CPB

duration correlated significantly to peak level and accumulated total of IL-8. Seven of nine ultrafiltrate samples contained IL-8 at levels similar to the plasma concentration. Changes in white cell counts were mainly attributable to neutrophils. The two subgroups did not differ significantly in neutrophil counts. Neutropenia found after 10 minutes of CPB was replaced by neutrophilia, with maximal values postoperatively. Calprotectin and IL-8 thus were released into the circulation during CPB in children. Ultrafiltration did not affect the plasma concentrations of these substances, and only IL-8 was detected in the ultrafiltrate.

L7 ANSWER 66 OF 68 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1995:333988 CAPLUS Full-text

DOCUMENT NUMBER: 122:129733

ORIGINAL REFERENCE NO.: 122:24135a,24138a

TITLE: Biomolecular mechanism of urinary stone and calcification and its similarity to that of atherosclerosis

AUTHOR(S): Kohri, Kenjiro; Itoh, Takaichiro; Hirota, Seiichi;

Umekawa, Tohru; Kurita, Takashi; Suzuki, Atsuo

CORPORATE SOURCE: Med. Sch., Nagoya City Univ., Nagoya, Japan

SOURCE: Saibo Kogaku (1994), 13(12), 1082-90

CODEN: SAKOEO; ISSN: 0287-3796

PUBLISHER: Shujunsha

DOCUMENT TYPE: Journal; General Review

LANGUAGE: Japanese

ED Entered STN: 04 Feb 1995

AB A review with 21 refs., on research history, composition anal., and formation mechanisms of kidney stone matrix, as well as the role of osteopontin, calprotectin, macrophage and other components in formation mechanism of kidney stone matrix.

L7 ANSWER 67 OF 68 MEDLINE on STN

DUPLICATE 22

ACCESSION NUMBER: 1993199399 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 8452432

TITLE: Calprotectin and complement activation during major operations with or without cardiopulmonary bypass.

AUTHOR: Garred P; Fosse E; Fagerhol M K; Videm V; Mollnes T E

CORPORATE SOURCE: Institute of Immunology and Rheumatology, National Hospital, Oslo, Norway.

SOURCE: The Annals of thoracic surgery, (1993 Mar) Vol. 55, No. 3, pp. 694-9.

Journal code: 15030100R. ISSN: 0003-4975.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199304

ENTRY DATE: Entered STN: 23 Apr 1993

Last Updated on STN: 6 Feb 1998

Entered Medline: 15 Apr 1993

ED Entered STN: 23 Apr 1993

Last Updated on STN: 6 Feb 1998

Entered Medline: 15 Apr 1993

AB Plasma concentrations of the granulocyte cell marker calprotectin were assessed during operation and 24 hours postoperatively in patients undergoing coronary artery bypass grafting with cardiopulmonary bypass, abdominal aneurysmectomy with implantation of an aortic graft, or thoracotomy without implantation of synthetic material. The concentration of calprotectin increased significantly ($p < 0.01$) in all three groups. Ten of the 30

patients in the group undergoing cardiopulmonary bypass received methylprednisolone at the start of the operation. No difference in calprotectin concentration was seen between the two subgroups ($p > 0.05$). Plasma concentration of calprotectin was shown to increase rapidly in patients undergoing cardiopulmonary bypass and aneurysmectomy, in whom complement activation also took place. However, the calprotectin concentration increased slowly during the operation and the postoperative period in patients undergoing a thoracotomy, in whom complement was not activated. At wound closure the calprotectin concentration was significantly elevated in the cardiopulmonary bypass and aneurysmectomy groups compared with the thoracotomy group ($p < 0.05$). The calprotectin concentration remained elevated during the postoperative period in all three groups. Our results indicate that calprotectin may serve as a suitable cellular marker when the biocompatibility of artificial surfaces is studied.

L7 ANSWER 68 OF 68 MEDLINE on STN

ACCESSION NUMBER: 1991369653 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 1892665

TITLE: Cardiac surgery and distribution of the leukocyte L1 protein-calprotectin.

AUTHOR: Semb A G; Gabrielsen T O; Halstensen T S; Fagerhol M K; Brandtzaeg P; Vaage J

CORPORATE SOURCE: Department of Physiology, University of Tromso, Norway.

SOURCE: European journal of cardio-thoracic surgery : official journal of the European Association for Cardio-thoracic Surgery, (1991) Vol. 5, No. 7, pp. 363-7.

Journal code: 8804069. ISSN: 1010-7940.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199110

ENTRY DATE: Entered STN: 8 Nov 1991

Last Updated on STN: 8 Nov 1991

Entered Medline: 24 Oct 1991

ED Entered STN: 8 Nov 1991

Last Updated on STN: 8 Nov 1991

Entered Medline: 24 Oct 1991

AB Activated polymorphonuclear leukocytes (PMN) secrete lysosomal enzymes, eicosanoids and toxic oxygen metabolites. In cardiac surgery patients, we measured arterial plasma levels of PMN and L1 (calprotectin), a prominent granulocyte protein, during cardiopulmonary bypass (CPB). The myocardial arterio-venous gradients were evaluated during reperfusion after cold cardioplegic arrest ($n = 10$). The arterial plasma concentration of L1 increased during CPB from 344 ± 71 micrograms/l (mean \pm SD) preoperatively to 5221 ± 1267 micrograms/l at the end of CPB (P less than 0.001). Simultaneously, the number of circulating PMN also increased (from $4.4 \pm 0.4 \times 10^9/l$ to $9.1 \pm 1.2 \times 10^9/l$ (P less than 0.05)). There was a positive correlation between the mean number of circulating PMN and the plasma level of L1 at all sampling times during CPB ($r = 0.93$, P less than 0.05). A coronary sequestration of both L1 (P less than 0.006) and PMN (P less than 0.002) was found after 5 min reperfusion. This was not present at 15 and 30 min reperfusion. The coronary entrapment of L1 and PMN did not covary significantly, and was unrelated to both the time of cardioplegic arrest and the arterial levels of L1 and PMN. In conclusion, the increased plasma concentrations of PMN and L1 during CPB and the coronary sequestration of both PMN and L1 may be factors in the pathogenesis of reperfusion injury of the myocardium.

=> d his

(FILE 'HOME' ENTERED AT 06:46:00 ON 11 OCT 2008)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 06:46:10 ON 11 OCT 2008
E SUNDREHAGEN E/AU

L1 155 S E3-E4

SET LINELENGTH 250 PERM

L2 2994 S CALPROTECTIN OR (L1(A)(ANTIGEN OR PROTEIN)) OR ((CALCIUM OR
CALCIUM-BINDING)(2A)MYELOID(2A)(8 OR 14 OR P8 OR P14)) OR

L3 7144 S CALPROTECTIN OR ("L1"(A)(ANTIGEN OR PROTEIN)) OR ((CALCIUM OR
CALCIUM-BINDING)(2A)MYELOID(2A)(8 OR 14 OR P8 OR P14))

L4 3693 S CALGRANULIN OR (MIGRATORY(A)INHIBITORY(3A)PROTEIN) OR MIF8 OR
MIF-8 OR MIF-14 OR MIF14 OR 27E10 OR L1H OR L1L OR S100A

L5 9967 S L3 OR L4

L6 118 S L5(25A)(HEART OR CVD OR ACS OR CORONARY OR CARDIAC OR CARDIO? OR
CARDIOVASCULAR OR ATHEROSCLERO? OR ARTERIOSCLER? OR

L7 68 DUP REM L6 (50 DUPLICATES REMOVED)

L8 1 S L1 AND L5

=> logoff hold

SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 06:53:49 ON 11 OCT 2008